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IMPORTANCE OF ORAL SURGERY TO PROSTHODONTIA

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THERE has existed for many years a great need for closer cooperation between the oral surgeon and the prosthodontist regarding surgical preparation and conservation of the dental ridge for dentures.

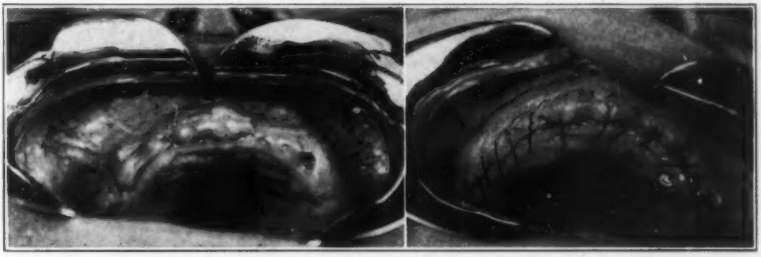

Fig. 1.—Maxillary ridge before (left) and after reduction of irregularities.

The prosthodontist is too often confronted with patients who have already had all their teeth removed and expect to have dentures constructed in mouths that are unfit for such a procedure. Every exodontist or dentist who removes teeth should leave the mouth in the best possible condition for dentures.[1]

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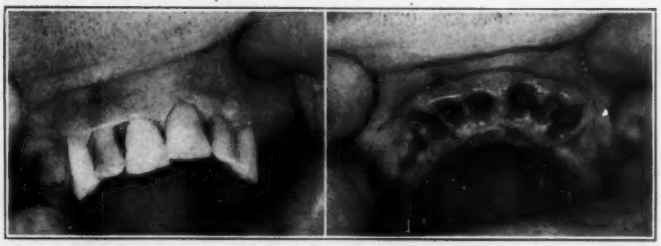

Fig. 2.—Left: Condition before extraction of maxillary anterior teeth. Right: Condition following extraction, showing protrusion of labial plate and evident need for reduction of this structure.

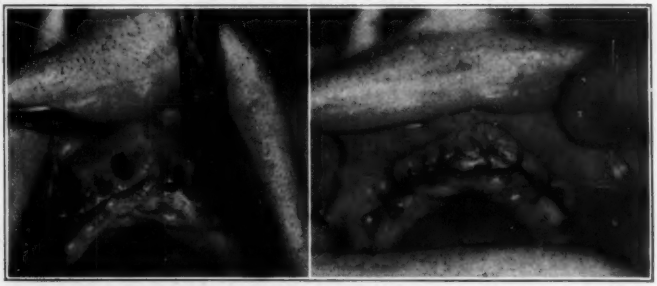

Fig. 3.—Immediate denture preparation. Left: Postextraction flap reflected to permit treatment of protruding irregular labial plate. Right: Flap returned to position and sutured loosely with continuous suture.

Fig. 4.—Left maxillary tuberosity contacting mandibular ridge when teeth are brought in occlusion. Treatment consists of reduction of the tuberosity.

The functional efficiency of correctly constructed dentures has always depended on the anatomic form and quality of the foundation upon which they must rest. (Fig. 1.) It is also true that dentures must be correctly

constructed in order to function properly. The oral surgeon should be just as concerned about the foundation on which the denture is to be placed as he is with the demands of the technic that enables him to obtain successful results.

Cogswell[1] says:

Fig. 5.—Prominent bulbous tuberosities before and after surgical reduction.

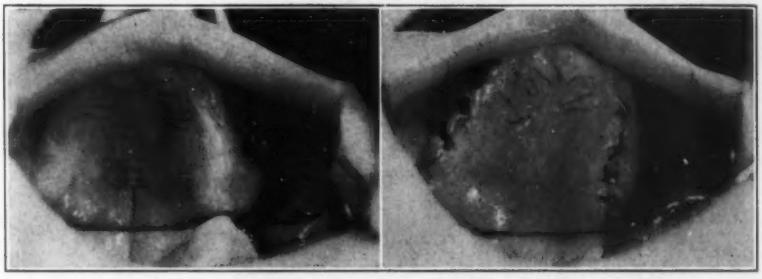

Fig. 6.—Protrusive left maxillary tuberosity before and after reduction.

The preparation of a mouth for dentures should begin, not at that period when dentures are inevitable and their need immediate, but at the time the first permanent tooth is lost. No tooth should be removed without consideration being given the possibility that the patient may eventually become e-

dentulous, and no tooth or teeth should be conserved if conservation may mean loss of the desired continuity and quality of the dental ridge tissues.

The labial and buccal section of the alveolar process should always be well reduced to remove undercuts and protuberances, and enough should

Fig. 7.—Retained maxillary third molars beginning to erupt after several years of full denture wearing. Routine roentgenography before bridge or denture construction would have located these. (Mirror view.)

Fig. 8.—Extensive bone destruction resulting from failure to find and remove the maxillary lateral incisor root fragment.

be cut away to make room for the denture base, to avoid a "pushed-out-lip" appearance in the finished denture. (Fig. 2.) The height of the

ridge is reduced, if necessary, according to the requirements of normal intermaxillary relations.

The longer the incision along the crest of the ridge, the horizontal incision, the less the necessity for cutting across the buccal face of the ridge, the vertical incision (Fig. 3), to free the flap without danger of tearing. Elevating the mucoperiosteum too much may cause formation of fibrous tissue, resulting in loss of fold. Lacerations in the form of accidental tears must be avoided. Impacted cuspids should be removed

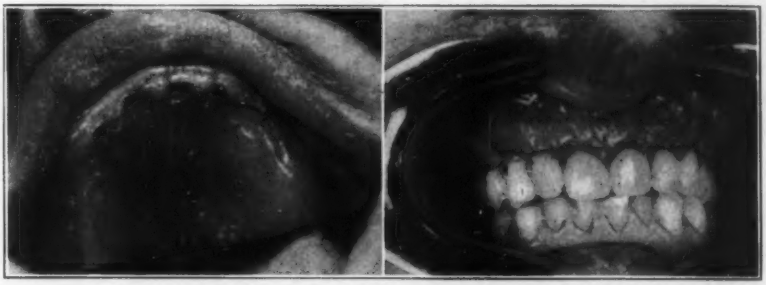

Fig. 9.—Redundant tissue which has grown over labial aspect of maxillary ridge as result of trauma on anterior part of restoration area. Left: Denture removed. Right: Denture in place.

Fig. 10.—Hyperplastic tissue under ill-fitting vulcanite denture. A large apron of redundant tissue covers the labial surface of the maxillary ridge. This may have grown from irritation of a loose denture. Its treatment is surgical removal. This condition may also be due to trauma from hammering of the natural mandibular incisors against the artificial maxillary teeth.

palatally by the elevation of a liberal palatal flap whenever possible. If they are removed buccally, it is difficult to obtain a peripheral seal for the denture owing to loss of structure and formation of fibrous tissue during the reparative process.

One area commonly overlooked is that of the tuberosity. Interference

may often be encountered where the ramus of the mandible may contact the maxillary tuberosity on the balancing side of the articulation, during a lateral or protrusive excursion of the mandible. (Fig. 4.) If interference is present, reduction of the tuberosity is necessary for the construction of a full maxillary denture, because this interference will not allow sufficient extension of the denture periphery in the hamular notch area. (Figs. 5-6.)

Undercuts in the retromolar fossae and those caused by the presence of the internal oblique ridge of the mandible will prevent extension of the lingual flange of the mandibular denture to its full limit. This extension is controlled by the function of the palatoglossus muscle, when the tongue is protruded. Usually, a reduction of the internal oblique ridge in this area is all that is necessary.

Root fragments and residual areas of infection are potential sources of sepsis. (Fig. 7.) It is the surgeon's obligation to eliminate such areas of infection before insertion of the dentures.

A routine full-mouth roentgenographic examination of an edentulous mouth is a precautionary measure for the prosthodontist. Much that is unsuspected may be exposed. (Fig. 8.)

Ennis[2] says that, in his experience, approximately 23 per cent of cases show retained roots, residual areas of old infection or impacted teeth.

The accompanying illustrations show what the prosthodontist sees when patients present themselves for denture service.

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Treatment of Osteomyelitis of Facial Bones with Penicillin.—The fundamental principles underlying the treatment of osteomyelitis of the facial bones are well illustrated by five cases. In contrast to the sulfonamides, penicillin prevents further spread of the infection, so that either before or after sequestration has occurred, devitalized bone can be removed surgically. Relapses are likely to occur until all the necrotic bone is gone. Surgical procedures are probably best postponed until the patient has had three or four weeks of penicillin therapy and sequestration has occurred, since there is then a better possibility of removing all the devitalized bone at one time. . . . The results in these five cases would seem to justify the hope that the present high mortality rate in cases of acute, subacute and chronic osteomyelitis of the facial bones will be drastically reduced when supplies of penicillin become generally available.—Kirby and Hepp, in *J.A.M.A.*, August 12, 1944.

SOME BENIGN SOFT TISSUE LESIONS OF THE ORAL CAVITY

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A PROBLEM of the average practitioner of dentistry is to differentiate small lumps and growths in and about the oral cavity. This material is not unusual, but is what one finds in routine practice. Because some similarity exists among these various anomalies, a brief review of their more common clinical appearance will be offered.

In the study of any neoplasm or other new growth, a careful examination is necessary to determine the physical characteristics. In addition, a microscopic examination of a section that includes adjacent normal tissue is an important precaution. A plan for study covers[1]:

1. The history of the tumor (time of appearance).
2. The precise location at the beginning.
3. The rate of growth.
4. The tissues involved.
5. The possibility of trauma as an exciting cause.
6. The condition of nearby teeth.
7. Pain, discomfort or disfigurement.
8. Glandular involvement (metastatic extension).
9. Evidence of any general systemic disease.

HYPERPLASIA OF SOFT TISSUE

Among the benign processes that occur in the oral tissues is hyperplasia of gingival tissues, which probably does not involve tumor formation in the strict sense of the term.

The term hypertrophy means an overgrowth of tissue in which the individual cells maintain their normal functions. The lesion has the appearance of a fold, which continues to grow in size. In some cases, there may be an excess of resilient tissue. Localized hypertrophy may be due to a mild chronic irritation. (Fig. 1.) Ill-fitting prosthetic appliances are the chief source of the condition in edentulous mouths, and this irritation permits atrophy of the alveolar ridge when dentures are not balanced properly.

Thoma[2] describes three types:

1. Granulomatosa: Granulation tissue growing from an extraction

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wound and due to continued irritation, from bony spicules or small sequestra.

2. Areolar hyperplasia: Folds of gingival tissue caused by wearing ill-fitting dentures.

3. Palatal hyperplasia: Hypertrophy of gingival tissue which results from the presence of suction chambers or vacuum cups in maxillary dentures. Histologically, the epithelium appears thickened as in small papillomas. (Fig. 2.)

The histologic picture usually presents squamous epithelium with ir-

Fig. 1.—Areolar hyperplasia caused by wearing ill-fitting denture.

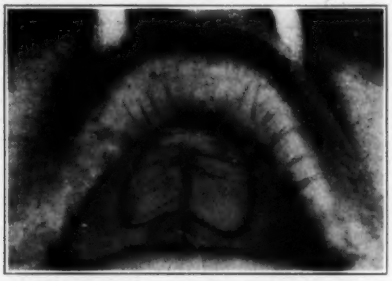

Fig. 2.—Palatal hyperplasia.

regular, broadened papillae growing into the corium. The corium is usually edematous and is composed of fibroblasts and collagenous fibers. At some points, there are keratinization and cracking of the papillary overgrowth. Sometimes, epithelial pearls are found beneath the epithelial surface.

Treatment.—After satisfactory local anesthesia has been established, the redundant flaps are lifted to produce tension on their attachment to the ridge. (Fig. 3, left.) The entire mass is resected away by the use of either electrocautery or a knife. This creates an area of perhaps 0.5 to

1 cm. of partially denuded periosteum and bone. (Fig. 3, right.) The use of sutures to approximate the tissue borders is not advisable as this would decrease the depth of the normal mucobuccal fold.

A satisfactory postoperative treatment consists of reinsertion of the patient's dentures after the addition of a small amount of compound on the periphery to act as a splint. The patient will be more comfortable if this material contains an obtundent. At twenty-four hour intervals, the dentures are removed, cleaned and inspected to determine whether they are

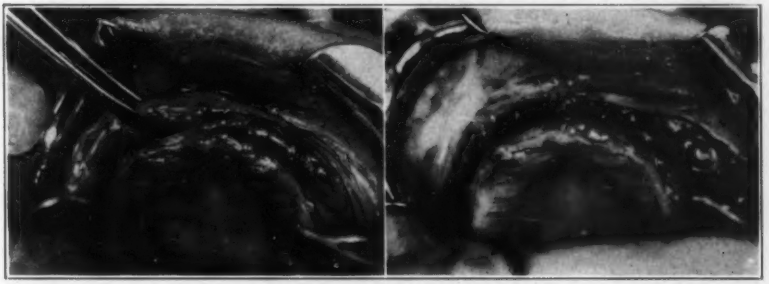

Fig. 3.—Left: Areolar hyperplasia (redundant flaps were lifted to produce tension on their attachment). Right: Incision for removal, which creates space of partially denuded periosteum and bone.

Fig. 4.—Leukoplakia on cheek.

holding the tissues in normal position. Healing is usually rapid, and new impressions can usually be made in two weeks.

LEUKOPLAKIA

Leukoplakia is characterized by grayish white keratinized patches on the mucous membrane of the body. In the oral cavity, the most common

location is the lips, gums, inner surface of the cheeks (Fig. 5), palate and tongue. (Fig. 5.) The lesion seems to be slightly sensitive. It occurs most commonly in men and is usually seen after the fourth decade. The lesion makes its appearance near a source of chronic irritation. Some lesions are

Fig. 5.—Leukoplakia on tongue.

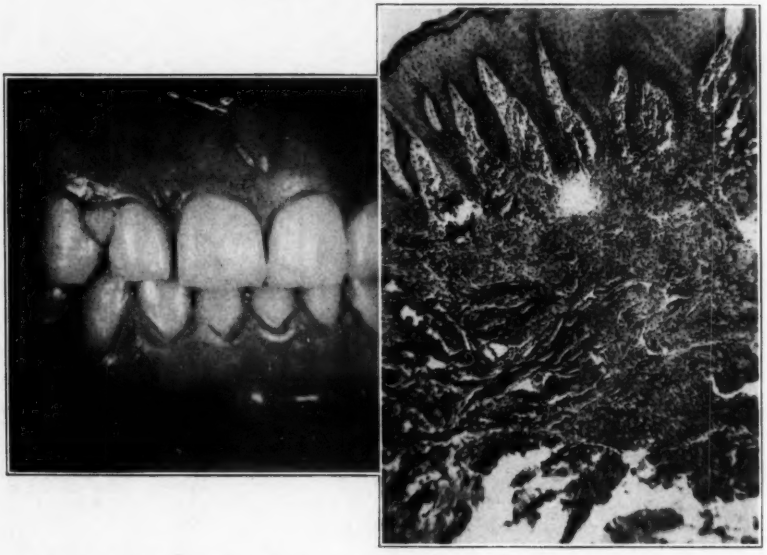

Fig. 6.—Left: Papilloma of gingiva. Right: Squamous epithelium with irregular, broadened papillae growing into corium. The corium, which is edematous, is composed of fibroblasts and collagenous fibers. At one point, there are keratinization and cracking of the papillary overgrowth, and several epithelial pearls are found beneath the epithelial surface.

believed to be due to systemic conditions, syphilis being the chief cause.[3]

The local causes are related to chronic irritation such as arises from the

use of tobacco, wearing ill-fitting prosthetic appliances, harboring carious teeth, poor occlusion and dental trauma.

The histologic section usually shows hypertrophic stratified squamous epithelium with an area of hyperkeratosis, acanthosis and atypical down growth rete pegs. Beneath the epidermis are scattered foci of lymphocytes

Fig. 7.—Left: Highly vascular peripheral giant-cell tumor. Right: Peripheral giant cell tumor.

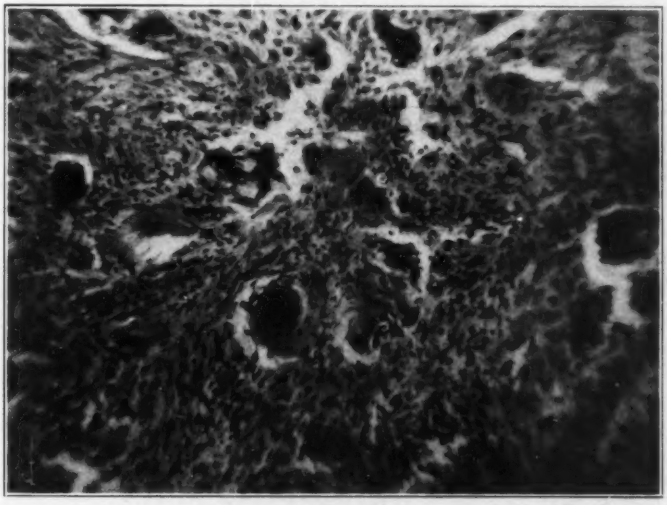

Fig. 8.—Benign giant cells. (High magnification of tumor, Fig. 7 right.)

and plasma cells in fibrous tissue. The epithelium is usually confined within its basement membrane.

Leukoplakia is a clinically benign lesion which may become malignant from constant irritation. The size of the lesion varies: it may be small or may extend over the whole dorsum of the tongue.

Treatment.—Treatment consists of (1) the elimination of all forms

of local irritation and chronic infection; (2) attention to systemic conditions, and (3) elimination of the lesion by electrosurgery, causing a scarring of the mucous membrane.

EPULIDES

From a clinical standpoint, a tumor which arises from the gum is known

Fig. 9.—Hemangioma, right buccal mucosa.

Fig. 10.—Fibroma of cheek.

as an epulis. This, however, is an unsatisfactory term because it may embrace a considerable variety of growths from the alveolar ridge attachment with a broad or narrow pedicle and a covering of normal epithelium. They sometimes undergo secondary ulceration due to chronic irritation, infection or pressure necrosis.

The most common growth of this type is the fibrous epulis (fibroma of the gums) and papillomatous type. (Fig. 6.) The fibrous epulis is painless and has a normal pink, smooth mucous membrane covering with a broad or narrow pedicle. It arises from the interproximal space and may be hard or soft. It is but slightly vascular. The growth is usually slow, changing little in months or even years.

The papillomatous type of epulis differs from the fibrous in that the epithelial covering is usually irregular or cauliflower-like. Both seem to develop in relation to a definite point of irritation. Both are clinically benign, but may become malignant from constant irritation over a long period of time.

Another form of so-called epulis is the peripheral giant-cell tumor. (Fig. 7.) The clinical characteristics of this type are a smooth, soft, cherry or

Fig. 11.—Fibroma of palate.

dark red nodular swelling on the gum surface arising from the interproximal space.

The peripheral giant-cell tumor may have a broad or narrow pedicle and its presence may cause a loosening or displacement of the adjacent teeth. Rapid growth is a nearly constant finding.

The roentgenogram of a giant-cell tumor reveals a thinning or rarefaction of bone at its source and, microscopically, immediately below the mucous membrane is a fibrocellular stroma. Scattered through this are numerous multinucleated giant cells. (Fig. 8.)

The finding of small masses of bone and cartilage has led occasionally to the classification of these lesions as giant-cell sarcomas. Such diagnoses are unfortunate because they lead to extensive tissue sacrifice, although the prognosis is usually favorable.

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Fig. 12.—Fibroma of gingiva.

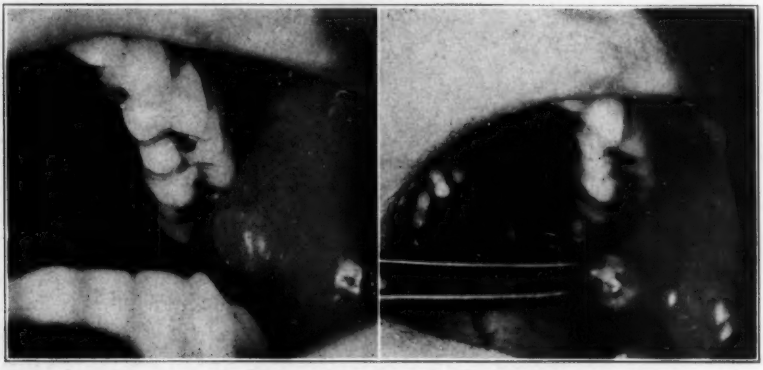

Fig. 13.—Left: Fibroma of cheek. Right: Dissected fibroma.

The best treatment for the fibrous and papillomatous type of epulis is a v-shaped incision, by a surgical or electrothermic method, into the submucosa. If all the tumorous mass is removed, the lesion does not tend to recur. All forms of chronic irritation should be eliminated.

Giant-cell tumors and all displaced teeth can be removed by surgical means. Also, when an area of bone destruction is present, removal and all necessary curettage is carried out. A precaution is cauterization of the

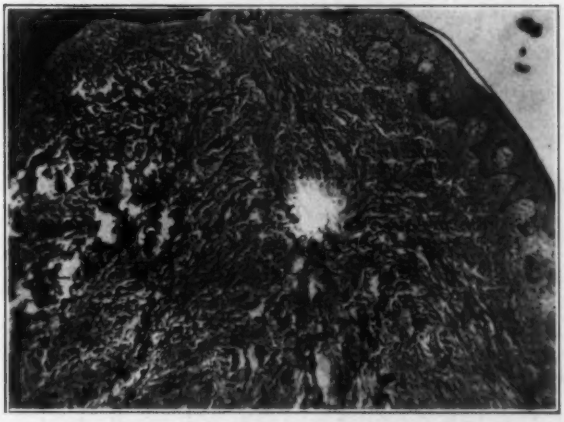

Fig. 14.—Photomicrograph of hypertrophied mucous membrane, ulcerated at several points. Beneath this, there is an area of fibrosis. The heavy connective tissue suggests an old scarring.

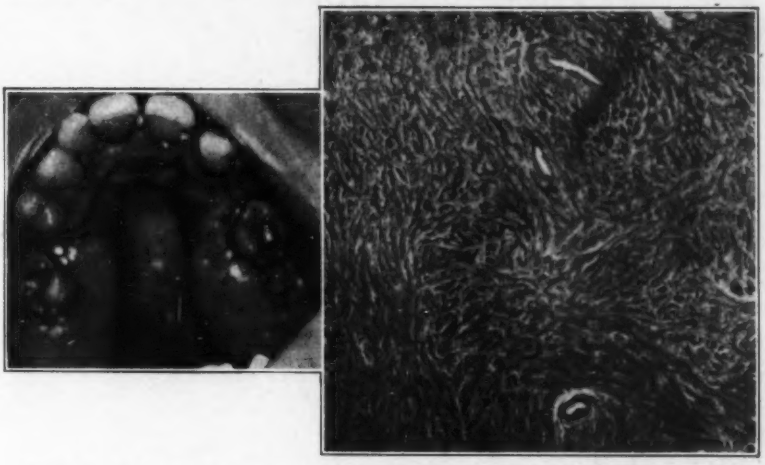

Fig. 15.—Left: Fibroma of tuberosity. Right: Photomicrograph of hypertrophied mucous membrane. Beneath this, there is a tremendous amount of scar tissue with a slight infiltration of pale mucoid tissue surrounding capillaries. The entire picture suggests a benign fibroma.

excised area with a chemical, actual cautery or irradiation, to prevent recurrence.

HEMANGIOMA

Hemangioma, a relatively rare tumor, is made up of an intricate mass

of capillaries and therefore bleeds freely if incised. It may or may not be congenital. It is purple-red, nodular, soft and spongy and is formed from the endothelium of blood vessels, probably of embryonic origin. (Fig. 9.) Hemangiomas may be found on the tongue, cheeks, lips and gingiva. They appear first as small purplish spots and grow to be large nodular projections.

Fig. 16.—Left: Adenoma near corner of mouth on right side. Right: Tumor elevated after partial detachment.

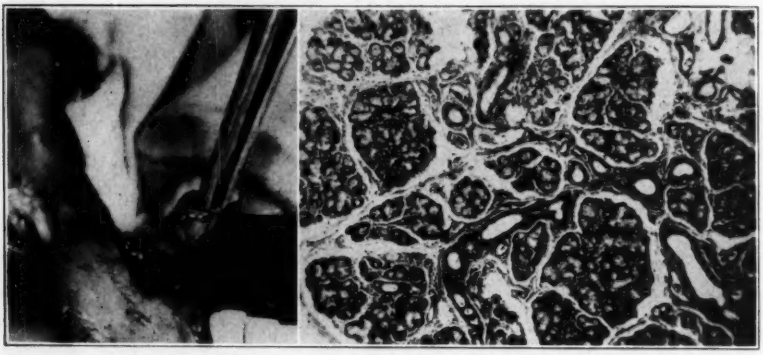

Fig. 17.—Left: Mass enucleated and elevated by tissue forceps. Right: Lobules of mucous-secreting acini embedded in loose edematous connective tissue. A cyst wall is lined by the same type of mucoid epithelium.

Though they are essentially benign, deaths have been reported from injuries that caused bleeding.

Surgical excision of the mass and tumor bed may cause considerable hemorrhage, requiring packing or cautery.

FIBROMA

Probably the most benign tumor found in the oral cavity is the fibroma (Figs. 10-14), a connective tissue growth arising from the deep layers of the mucosa or from the periosteum. It may develop from the terminal stage of an inflammatory hyperplasia, as does the epulis granulomatosa.

The fibroma, which has either a broad or a narrow base, is usually circumscribed and well defined and is covered with normal membrane. There are two types, the hard and the soft. They grow slowly and may even be dormant for years. Usually, they are small, the average being 6 - 8 mm. in diameter. It may be larger. Fibroma, which seems to be the result of chronic irritation, occurs on the gingiva, cheek, lip, tongue and hard and soft palates. Those that arise in the interproximal spaces are usually classified as epulides. Fibroma causes no discomfort, but may interfere with

Fig. 18.—Mucocele on lip.

speech and mastication. If the source is the periosteum, interproximal bone distention seems to result from this surface. If the tuberosity is the site of the growth, prosthetic work is complicated. (Fig. 15.)

Chronic irritation of a fibroma causes ulceration, which promotes growth, with invasion of the underlying tissue, which transforms the benign tumor into a malignant neoplasm.

ADENOMA

The adenoma is a soft tissue tumor resembling in structure a secreting gland. (Figs. 16-17.) It is commonly situated on the oral mucosa. From a clinical standpoint, it resembles a retention cyst or mucocele. (Fig. 18.) It

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may expand or invade bone when situated near it. Roentgenographically, this process is indicated by an area of radiolucency outlining the tumor.

Histology.—Histologically, the process appears as an irregular organization of mucous gland structure, usually encapsulated.

Treatment.—Surgical enucleation of the mass by making an incision across the body of the membrane and elevating the tissue by use of forceps, dissecting around the periphery, is the treatment of choice. The tumor bed should be cauterized by chemical or electrosurgical methods.

RANULA

Ranula is a general term applied to a chronic benign cyst that forms in the floor of the mouth, owing to the obstruction of a duct of a mucous or

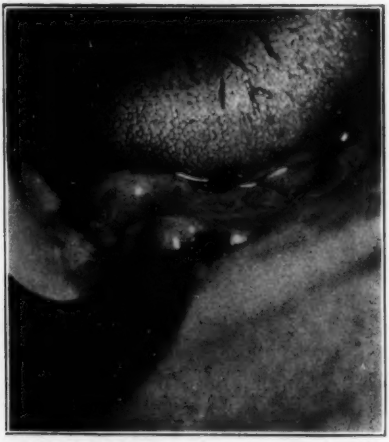

Fig. 19.—Ranula.

salivary gland, usually from the sublingual gland, rarely, if ever, from the submaxillary. The ranula forms as does a mucocele, but develops to a larger size. (Fig. 19.) The typical ranula is located under the tongue, lying above the mylohyoid muscle attachment. It is of varying dimensions, depending on its age or rate of growth. There is no pain or tenderness, except when the tongue is raised, and this restriction interferes with speech and mastication. The fluid content may be easily palpated and the cyst wall is subject to rupture when injured. The ranula contains a sticky, mucoid fluid.

Roentgenographic examination after the injection of a lipiodol solution reveals the size and course of the cavitation and aids in differential diagnosis between a tumorous mass and a cystic cavity.

Treatment.—The treatment consists of (1) complete excision of the

cyst, which may be technically very difficult because of the extremely thin wall, and (2) removal of the upper wall and the superimposed membrane and suturing of the incised mucous membranes to the margin of the cyst wall. This makes the cyst a part of the mouth cavity and healing is reasonably certain.

SUMMARY

The occurrence of benign, easily managed growths about the oral cavity is far more common than is malignancy. Hence, the ability of the general practitioner to identify these lesions as benign constitutes a safeguard for the patient. Their recognition and early conservative treatment will do much to prevent unnecessary worry and occasionally needless loss of tissue or even mutilation.

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Chemicosurgical Treatment of Cancer of the Lip.—The chemosurgical method was introduced by Mohs for the excision of accessible forms of cancer under microscopic control. The tissues in question are fixed *in situ* by means of a suitable solution of zinc chloride and then excised and examined microscopically. This process is repeated if necessary until microscopically cancer free surfaces are reached. The fixation of the tissue by the zinc chloride does not interfere with the microscopic work. . . . The cure rate of 87.5 per cent after five years appears to surpass recently published rates for surgical and radiotherapeutic methods of treatment of cancer of the lip, but of course these rates are not based on sufficiently standardized material to yield strictly comparable results. This is particularly the case with respect to radiotherapy by standardized methods in expert hands. There would seem to be no question, however, as to the value of the chemosurgical method. The microscopic control of excision gives the method an unequaled advantage—"time after time, unsuspected outgrowths of small caliber from the main tumor mass were found microscopically, at times extending a considerable distance after becoming grossly invisible." The method is practically without risk, it conserves lip tissue and it has no tendency to cause metastasis. As a rule regional metastasis must be removed by standard surgical dissection. "Chemosurgical treatment is particularly advantageous for cancer of the lip recurrent after surgical operation or irradiation. Such lesions often respond poorly to repetition of the original therapy, but they almost invariably respond to chemosurgical treatment."—*J.A.M.A.*

CYSTS OF THE JAWS

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A CYST is any sac, not necessarily pathologic, but a cyst of the jaws implies bone destruction with resulting cavitation of varying extent, varying from a small apical area above a maxillary incisor to a massive space occupying a great portion of the maxilla or the mandible.

A general classification of true cystic tumors of the jaws can be made as follows: (1) radicular and root-end cysts; (2) dentigerous or follicular cysts; (3) traumatic cysts, and (4) adamantinomas (ameloblastomas).

Waldron[1] classifies this entire group as epithelial odontomas.

Fig. 1.—Granuloma of maxillary left lateral incisor.

The radicular cyst is usually found in the apical areas of pulpless teeth. It may follow pulp death, trauma or failure to remove residual lesions at the time of extraction. The epithelial granuloma (Figs. 1 and 2) is a potential cyst, and provision must be made when the tooth is removed to permit thorough exposure and enucleation of all of this tissue. In multi-rooted teeth, excision of bony septa facilitates this; whereas, in the anterior part of the mouth, a semilunar flap and an apical window provide excellent access. Extraction of the diseased tooth alone is not sufficient. (Fig. 3.)

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Schlack[2] says that 41.7 per cent of granulomas contain epithelial rests.

Kazanjian[3] says that dental root cysts are caused by chronic irritation at the apex of devitalized teeth. Examples of these are the huge radicular cysts (Fig. 4), which develop above pulpless maxillary lateral incisors and destroy all but a small portion of the supporting bone of the teeth. This

Fig. 2.—Granuloma of maxillary first molar; showing antral shadow superimposed.

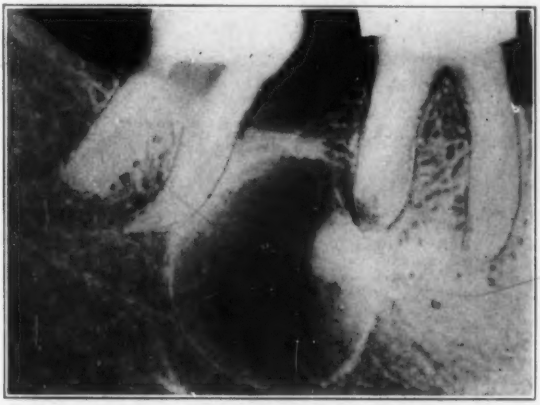

Fig. 3.—Large cyst of mandibular pulpless molar; showing how little drainage would be provided by extraction of tooth.

region of the maxilla has a thin labial plate and is particularly susceptible to this type of expansive bone destruction.

Cyst formation, from the observations of Kronfeld,[4] seems to follow breaking down of the granuloma into a serous fluid in which epithelial

remnants form inside a fibrous membrane. This growth causes peripheral pressure, which initiates formation of a thin layer of cortical-like bone around the new development area.

The dentigerous or follicular cyst (Figs. 5-6) arises from the irritation of an unerupted or an unerupted and impacted tooth. It is believed to develop as a result of some pathologic change in the enamel organ of the developing tooth and so it is entirely epidermal. The dentin of the crown

Fig. 4.—Pulpless maxillary left lateral incisor root end extending into cystic cavity; showing small amount of bone ridge that remains.

Fig. 5.—Cyst, probably follicular, surrounding third molar crown, causing extensive bone destruction, particularly of distal surface of second molar.

is of mesodermal origin and is usually the only part of the tooth in the cystic cavity. The fibrous wall of the cyst is lined by epithelium derived from the peripheral layer of cells of the enamel organ.[4]

Traumatic or extravasation cysts are hemorrhagic in origin and follow injury that has caused bleeding into the tissue. They are found on exposure to be cavities within bone filled with blood, but there is no lining membrane.

The adamantinoma contains the essential enamel-forming organ. It is invasive and its treatment is more radical. Ivy says resection of the jaw is necessary to prevent recurrence.

CHARACTERISTICS OF CYSTS

Cysts (1) grow slowly; (2) form a definite cavity, which, in the early

Fig. 6.—Left: Dentigerous cyst of mandibular right third molar; showing the extension to distal aspect of second molar. Right: Condition after removal. Normal regeneration is taking place.

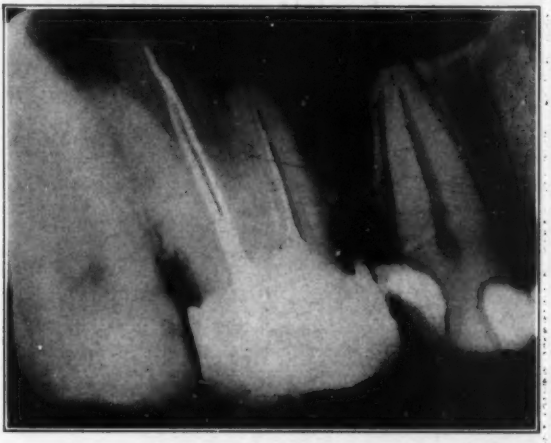

Fig. 7.—Low antral floor. Between the molar and bicuspid, the inferior border of the antrum reaches almost to the gingiva.

stages of development are surrounded by healthy bone; (3) are usually attended by no discomfort unless the fluid in the cyst cavity becomes infected; (4) may assume a large size; (5) weaken the jaw; (6) are benign; (7) because of extension are, on removal, not uncommonly followed by paresthesia of the lower lip, and (8) if bone destruction has been slight,

may cause a fracture, on trauma, and, if incompletely enucleated, may recur.

DIFFERENTIAL DIAGNOSIS

Even the very early essays on dental radiography dwelt at length on the danger of mistaking normal radiolucent areas in the mandible or in the maxilla, a portion of the antrum, for a cyst. In spite of these admonishments over the years, maxillary teeth sometimes are still sacrificed when their root ends lie close to an antrum, the floor of which dips low (Fig. 7),

Fig. 8.—Massive cyst of left maxilla resembling maxillary sinus variation in Figure 6.

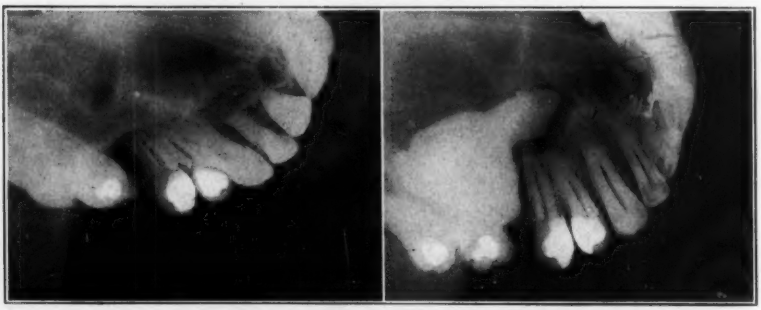

Fig. 9.—Left: Antral nasal fossa and cystic shadows, all seen, but not differentiated clearly. Right: Injection of lipiodol outlining clearly periphery of cyst.

and a normal antrum is occasionally opened into because of its cystic appearance.

To avoid such errors, there are several safeguards: (1) adequate roentgenography; (2) puncture biopsy; (3) injection of a radiopaque fluid, and (4) careful digital examination.

Ennis has frequently shown how correct anteroposterior or occlusal views will locate accurately and outline an obscure maxillary cyst. His findings prove that competent roentgenography will reveal much that insufficient roentgenography leaves in doubt.

The insertion of a needle into a suspected cyst and the aspiration of cystic fluid will settle any previous doubt.

When a cyst lies close to the maxillary sinus and encroaches upon its floor or other boundaries, and no sharp line of demarcation is apparent,

Fig. 10.—Cystic condition around crown of impacted mandibular right molar. The patient experienced no discomfort until contents became infected.

Fig. 11.—Massive dentigerous cyst of mandible (left view) that apparently has grown from unerupted and impacted left cuspid tooth.

the injection of lipiodal followed immediately by roentgenography will yield the desired information. (Figs. 8-9.) A fistula, if present, may be used for introducing the solution, but if not, penetration through the cyst wall is simple.

UMI

Careful digital examination will yield much information. In many cases, some swelling is present and if, on light pressure, this is at all palpable and in addition there is a crackling suggesting parchment, the probability that a cyst is present is very great.

SYMPTOMS

Cysts do not as a rule cause pain and discomfort. Until acute distress begins from suppuration (Fig. 10), the patient is often completely unaware

Fig. 12.—Right view.

Fig. 13.—Postero-anterior view.

of its presence, but, when it is large (Figs. 11-13), the facial contours may be changed and, intra-orally, the alveolar process or palate may show a smooth enlargement. Some pain of a referred nature from pressure or a nerve trunk is sometimes present.

TREATMENT OF CYSTS

After diagnosis has been established and the position and size of the

cyst have been determined, treatment consists of exposure, drainage and partial or complete enucleation of the membrane. After sufficient exposure by an incision, an attempt should be made, by using a flat broad instrument, to separate the sac from its bony environment without rupturing it. (Figs. 14-16.) This is much easier while the cyst maintains a bladder-like form than after its collapse, and clearer vision of adjacent structures is possible.

However, after puncture, the contents can be drained and, by tension on the loosened portion, the remaining attachment is separated. Rough manipulation can cause damage in either jaw. In the mandible, if expan-

Fig. 14.—Left: Intra-oral appearance of cyst; showing bulbous protrusion of soft tissue above maxillary second bicuspid. Right: Incision of overlying soft tissue.

Fig. 15.—Left: Cystic membrane loosened from surrounding bone. Right: Cystic sac partially enucleated.

sion of the cyst has reached the canal, the separation of cystic membrane from the blood vessels and nerve may be difficult and needs to be carried out with extreme care. Tearing and unwise incising are certain to be harmful.

In the maxilla, the maxillary sinus and nasal floor are the hazards. Here, in large cysts exposed by extensive incision, a good plan is to incise the cyst wall horizontally and, after aspirating the contents, examine all walls with a blunt instrument for soft areas, which indicate a membraneous separation

only from the antrum or nasal cavity. The presence or absence of such spots obviously determines the treatment to be followed.

The Partsch procedure is based upon this principle of incision and drainage of the cyst and of maintenance of adequate communication with the oral cavity, of which the cyst cavity becomes a part. In every case, adequate exposure of the cystic cavity is necessary and a window must be maintained throughout healing. Occasionally, in large cysts of the palate (Fig. 17, left),

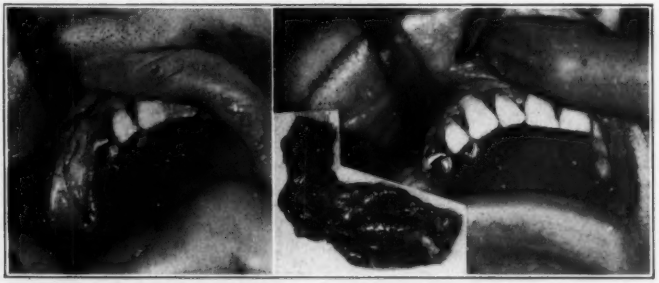

Fig. 16.—Left: Cystic sac completely enucleated. Right: Bone cavitation after removal of cystic membrane Inset: Gross appearance of cyst after removal.

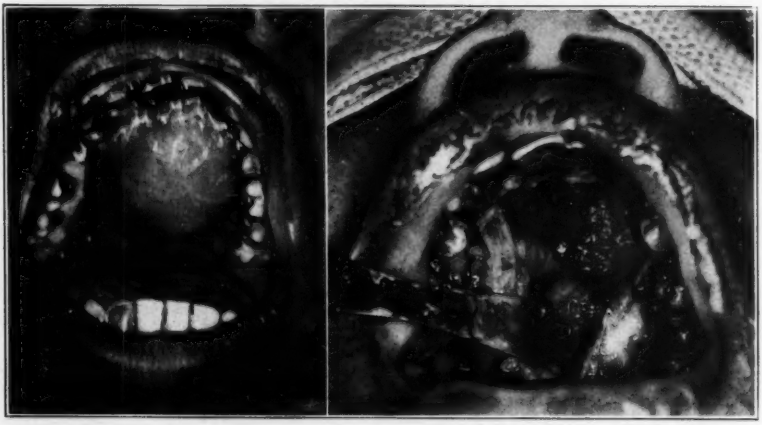

Fig. 17.—Left: Large cyst of palate that had displaced nasal and antral floors. Right: Palatal flap reflected and cystic membrane exposed.

proper exposure is possible only by laying back most of the palatal mucoperiosteum. (Fig. 17, right.) Then, when the task of the cystic membrane removal is complete, the flap is returned to position and sutured. Now a labial window (Fig. 18) is made to permit treatment and inspection of the cavitation as long as is necessary.

This provides a means of covering a nasal or antral perforation that may have been caused by the growth or by operation.

SUMMARY

The bone-destroying tendency of a cyst is particularly noticeable in alveolar bone. Here, but a few millimeters of ridge periphery may remain

Fig. 18.—Labial window for removal of remaining membrane and for inspection and treatment while healing progresses.

Fig. 19.—Roentgenographic appearance of cyst of palate.

around the teeth and the promiscuous extraction of involved or suspicious teeth at the time of cyst removal can carry this away and leave a deformity, which will needlessly complicate prosthetic work. Therefore, the extraction of teeth must be delayed until sufficient bone has regenerated to permit it without mutilation.

Also, far too many teeth are sacrificed needlessly in cyst treatment,

either to create an approach, or because the operator does not know that temporarily pulpless and displaced teeth will regain normal qualities and position after healing. Not infrequently, massive cysts originate from one tooth only, and even this, if difficult to replace and single rooted, may be retained through root canal surgery and, if necessary, root resection.

CONCLUSIONS

1. Correct diagnosis of a small or an obscure cyst is sometimes difficult, but can be established by standard methods.

2. Errors in diagnosis are unfortunate for the operator, but more so for the patient, who loses useful teeth and undergoes surgical operation because of the mistake.

3. Teeth the apices of which extend into cystic cavities and are displaced in the arch and react negatively to vitality tests should not be condemned

Fig. 20.—Photomicrograph of cyst of palate.

and removed at the time of cyst treatment. Many will regain their normal position and vitality, as healing progresses.

4. The point of entry into the cystic cavity is important, to avoid damage to frail strands of peripheral bone. These may constitute the only remaining osteogenic sources and their conservation can reestablish ridge normalcy, whereas their loss could mean local deformity.

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OSTEOFIBROMA OF THE MANDIBLE: REPORT OF A CASE

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THE relatively rare benign osteogenic tumor, osteofibroma, occurred in a young and healthy man, and, because of its rather similar appearance, was treated as an osteomyelitic lesion with necrosis. Differentiation by a roentgenogram from such a condition is not easy, for osteofibroma presents a moth-eaten osseous appearance also, and the seemingly walled condition of the central mass hints at imminent sequestration.

There is, however, a marked dissimilarity from an osteogenic sarcoma, the common malignant invader of the jaws. The latter differs in that the lines of demarcation are absent. Instead, infiltration is the rule, and no division is apparent between the normal bone and the neoplasm.

REPORT OF CASE

History.—D. E. G., S.C. 3/c, aged 20, born in the United States, was referred to the Naval Dental School for consultation. One year prior to admission, he noticed a swelling in the left mandibular first molar region, tender to the touch.

In August 1943, he went to a dental dispensary for consultation. The primary diagnosis was osteomyelitis. He was treated for a few months for this, but recurrent pain and tenderness continued.

The father was tuberculous. The mother and only brother were living and well.

Examination.—The patient was well developed, weighing 200 pounds. The Kahn and Wassermann tests were negative, as was urinalysis. The blood pressure was 120/84. The temperature was normal. Oral examination revealed a hard semispherical nodular immobile, palpable mass 4-5 cm. in diameter in the left mandibular first molar region. It was covered with normal mucosa. The mass seemed firmly attached to bone. There was marked asymmetry of the face on the left side. There was no adjacent adenitis. The molar responded to vitality tests. A presumptive diagnosis of osteoma was made.

Roentgenographic Examination.—An intra-oral roentgenogram showed the roots of the mandibular first molar extending into an area of bone change. (Fig. 1.) The apices had been lost through absorption. This new bone forma-

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tion was of varying degrees of density and was surrounded by a narrow strip of radiolucency that appeared to separate it from the normal bone.

In the occlusal view of the growth, there was an extreme bulging of the buccal plate of the mandible, with marked loss of density between the first and third molars. (Fig. 2.)

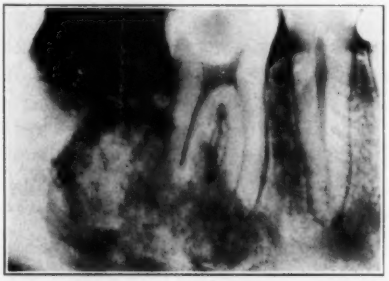

Fig. 1.—Intra-oral view of tumorous mass and involved mandibular first molar.

Fig. 2.—Occlusal view of osteofibroma.

Operation.—The gingival tissue was separated at its attachment from the first bicuspid posteriorly to the distal aspect of the first molar and an incision was made along the crest of the ridge distally for 3 cm. A corresponding flap was laid back to expose the lateral surface of the mandible.

Pressure from the application of molar forceps on the tooth moved the entire mass slightly and a bur was used to complete the separation. The tooth and attached growth were then carefully lifted out.

The bone defect that remained was very large, and even light traction on the mandibular incisors caused the parts on each side of the cavitation area to move independently. There was no fracture, but a small portion of cortical bone remained.

The flap was returned to position and healing was normal and without pain except for mild discomfort from bone exposure, which was readily relieved with gauze dressings.

Gross Examination.—The specimen consisted of a hard, bony line, irregularly shaped tumor mass measuring approximately 5 cm. in diameter and

Fig. 3.—Mandibular first molar roots embedded in tumor mass.

firmly attached to the left mandibular first molar. (Fig. 3.) The section did not seem to be completely encapsulated.

A small section of the outer layer of the gross specimen was referred to the pathologist. The preliminary microscopic examination showed fragments of bone and connective tissue in which there was old blood pigment. There are a few giant-cell osteoclasts. Most of the bone spicules were eroded and undergoing degeneration. There was some fresh hemorrhage. Overlying this were spicules of new bone surrounded by osteoblasts embedded in vascular connective tissue. The primary pathologic diagnosis was ossifying hematoma.

The large tumor mass was decalcified and embedded in celloidin, and the

sections were stained with hematoxylin and eosin. Microscopic examination revealed that the tissue was composed of fine bony trabeculae with fibroblastic marrow. (Fig. 4.) The relative amounts of marrow and trabeculae varied in different regions. There were large islands of purely fibrous tissue with newly formed trabeculae. Many large foreign body giant cells were present. The bony mass was surrounded by fragments of vascular connective tissue. There was no sign of mitosis. On the basis of further tissue study, a diagnosis of osteofibroma was made.

Because of failure to identify them as definite entities and by reason of their undeniable resemblance to some well-established diseases of bone, these growths have been given many names, among which are osteofi-

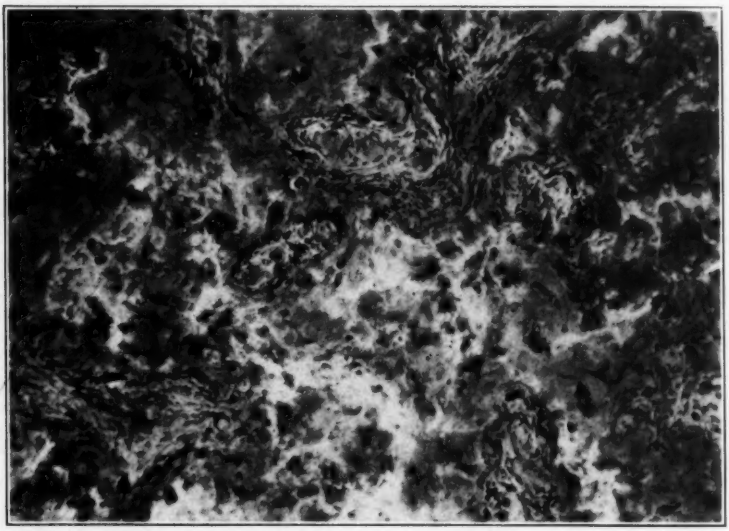

Fig. 4.—Section from center of osteofibroma, showing fibrous stroma in marrow spaces and new bone formation.

broma, ossifying fibroma, osteoma and fibrous osteoma. The process has been referred to as exostosis and localized osteitis fibrosa.

There are certain findings that are, in general, common to all[1]:

1. There is no bone disease in the remaining skeleton.
2. No cases have been reported to have undergone sarcomatous change.
3. There are no abnormalities of the blood calcium and phosphorus.
4. The lesions are benign, yet the condition may recur and require further resection.

Potts and Hatton,[2] in reporting a case, state that the bony tumor mass resembles that of a calcified fibroma.

Geschickter and Copeland[3] say, "These tumors can be distinguished from the osteogenic sarcoma by the absence of malignant nuclei and atypical mitotic figures in the spindle cells and osteoblasts."

Dewey[4] shows preoperative roentgenograms and postoperative photographs of a condition very similar to this one. The mass appeared walled off in a like manner and, after removal, its appearance resembled that presented in Figure 3.

Thoma[5] agrees in general with previous writers on the subject. He believes that these tumors are more common in the maxilla than in the mandible and that they occasionally occur simultaneously in the two jaws.

SUMMARY

Correct diagnosis is imperative in the examination of lesions of the maxillary bones. To interpret an osteofibroma as malignant and then to treat it as such, with radical methods, is certain to result in mutilation.

Furedi[6] says, "The conclusion of our clinical observations suggests to us that the indication for surgery has to be decided upon individually until our knowledge concerning the etiology is much more definite and clear, when other treatment may suggest itself. These lesions should never be operated upon as neoplasms; in other words, never radically." Similarly, a diagnosis of osteomyelitis for osteofibroma will be disappointing as well. Neither the provision for drainage nor the long wait for sequestration will be rewarded, but instead the tenderness and increase in growth will both persist.

The report of finding a giant cell by the pathologist or of the semblance of sarcoma is not enough to excuse hasty or extensive surgical operation on neoplasms in the maxillary bones. There are factors in this seeming likeness to malignancy such as more or less than average density of bone, imperfect roentgenography and an incomplete or indefinite history of its development. However, by careful evaluation of clinical findings and the fullest use of known diagnostic aids, the differentiation of osteofibroma from other lesions of the jaws is reasonably sure.

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UMI

PROSTHETIC APPLIANCE FOR COMMINUTED FRACTURE OF THE NOSE: REPORT OF A CASE

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A man, aged 22, in good physical condition, presented himself for examination after sustaining traumatic injuries to the face and nose. Clinical examination revealed that the nose was badly displaced, and there was subcutaneous hemorrhage of the frontal region of the face, with slight nasal bleeding.

Fig. 1.—Lateral study of facial bones showing complete transverse fracture through base of nasal bone approximately 2 cm. back of tip of bone.

Lateral and postero-anterior roentgenograms of the facial and nasal bones revealed a complete transverse comminuted fracture through the base of the nasal bone approximately 2 cm. back of the tip. (Fig. 1.)

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Fig. 2.—Split button cast gold splint on dental cast, secured by wiring in bicuspid-molar regions.

Fig. 3.—Relationship of splint, binding posts, connectors and acrylic nasal splints.

Use of a headcap and an external nasal appliance that would be secured to the bridge of the nose was impossible owing to the comminuted condition of the nasal bone. The use of an external appliance attached to a headcap was deemed inadvisable because of lack of traction on the comminuted nasal bones and the probable occlusion of the nasal fossae.

It was decided to use a cast gold intra-oral splint as described by Col. Roy Stout (U.S.A.), which was made from the model of the maxillary arch. To the anterior part of this splint was added metal binding posts (three sixteenths inch screw top—radio type). (Fig. 2.)

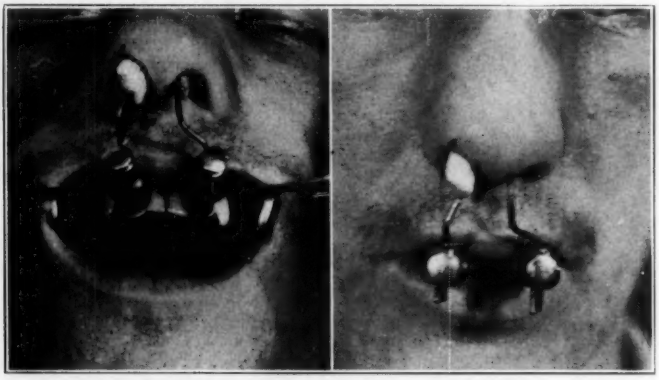

Fig. 4.—Left: Connectors contoured so that acrylic splints are in proper position and firmly secured. Right: Appliance in place four days after reduction of fracture.

The next step was the construction of acrylic nasal splints made from an original beeswax impression of normal nasal fossae. Stainless steel rods were embedded in the inferior end of these splints to serve as connectors to the binding posts on the cast splint. (Fig. 3.)

The cast gold splint was secured to the teeth by interproximal wiring before an attempt was made to reduce the fracture.

Under pentothal sodium anesthesia, the fracture was manually reduced and the acrylic splints were inserted in proper position with any further adjustment of the steel connectors considered necessary. (Fig. 4.)

Roentgenographic examination made after the application of the appliance revealed satisfactory reduction. (Fig. 5.)

COMMENT

The appliance was worn three weeks, during which the patient had but slight discomfort and was able to perform light duty. The reaction of the nasal mucous membrane to pressure was constantly observed and, at the end of a three weeks' period, there was no apparent tissue change and normal airways had been maintained.

This type of appliance for treatment of comminuted nasal fractures has a distinct advantage over the plaster headcap in that it affords maximum immobility and a minimum of discomfort without the obvious disadvantages of a headcap.

Fig. 5.—Immobilization apparatus in place, attached to upper teeth. There is an oblique fracture through one or both sides of the distal end of the nasal bone, but no appreciable displacement is seen.

CONCLUSION

This method offers a simple yet practical means of treating comminuted fracture of the nasal bones by the use of a dental prosthetic appliance. The maxillary arch bar does not harm the teeth or tissues and its application causes no disability. The patient may continue his usual duties.

UMI

ACTINOMYCOSIS TREATED WITH CRUDE LYMPH GLAND EXTRACT

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TRAUNER,[1] in 1930, described a method of treating actinomycosis in which an alcoholic extract of the cervical lymph glands of cattle was applied on dressings directly to the diseased tissues as an irrigant and by subcutaneous injection. Successful results are reported by Trauner[1] and Kotanyi.[2]

The unusually favorable outcome of two cases of long-standing actinomycosis treated with an aqueous lymph gland extract at the Dental Clinic of West China Union University merits consideration. An aqueous extract was used to avoid pain ensuing from contact of denuded tissues with alcohol. Lymph glands from goats were substituted for those of cattle to preclude the transmission of tuberculosis.

PREPARATION OF THE EXTRACT

Fresh glands were immersed in 75 per cent grain alcohol for fifteen minutes to sterilize the outer surfaces. The capsules were then removed under aseptic conditions, and the glands were cut into small pieces and crushed and ground in a mortar. Physiologic sodium chloride solution was added from time to time during grinding until a semiliquid mix resulted. The extract was filtered through a piece of fine gauze and preserved in a tightly stoppered bottle containing a drop or two of carbolic acid. The solution was kept in a cool place when not in use. Extract more than 3 days old was discarded.

REPORT OF CASES

CASE 1.—*History.*—A farm girl, aged 18, appeared at the Dental Clinic April 20, 1942, complaining of stiffness in the left jaw, first noticed a month earlier, and inability to open the mouth. The temperature was normal. The cervical lymph glands on the affected side were not enlarged. A tentative diagnosis of pericoronitis of a left mandibular molar tooth was made. Treatment brought no relief after three days and the patient discontinued visits to the clinic.

December 3, she again presented herself for treatment, this time with greatly exaggerated symptoms.

Examination.—Although there was little pain, the left side of the face, including the preauricular, infra-orbital and mandibular regions, was red, swol-

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len and indurated. The temperature was 99.2° F., pulse 80, white cell count 9,000 and urine negative. The submaxillary lymph glands appeared slightly enlarged. Extra-oral roentgenograms disclosed no abnormality of the temporomandibular articulation or bony structure of the jaws.

Treatment and Course.—The affected area was irradiated with doses of about 100 r, one-third of the erythema dose at the kilovoltage employed, twice weekly for two weeks, with some reduction of swelling and trismus. Sulfanilamide and sodium bicarbonate, 1 gm. each every four hours, a warm saline mouth wash and hot wet dressings applied for thirty minutes every three hours were prescribed for four days following discontinuation of the x-ray therapy, without appreciable effect. An exploratory operation in the third week revealed no pus in the parotid capsule.

Ten days after operation, five small (2 by 4 cm.) closely grouped purple abscesses developed in the preauricular region. They yielded a yellow pus containing many tiny sulfur-colored granules.

Diagnosis.—Microscopic examination of the granules disclosed typical flower-like colonies of the actinomyces, identification of which was confirmed by culture. On establishment of the diagnosis, the patient admitted a habit of picking her teeth with straw.

Operation and Aftercare.—The abscesses were incised and drains of rubber dam inserted. Potassium iodide was administered for a week: 10 grains three times daily for three days and 30 grains three times daily thereafter, without satisfactory improvement.

January 15, 1943, the lesions were irrigated with fresh lymph gland extract twice daily. The extract was further prescribed as a mouth wash in an aqueous dilution of 10:1.

Outcome.—Ten days after the institution of treatment with the extract, no actinomyces could be found in the discharge or on the dressings. The patient was dismissed completely cured March 2.

CASE 2.—*History.*—A man, aged 25, a clerk, entered the dental clinic June 11, 1943, complaining of stiffness of the jaw and presenting a firm purple-red swelling over the angle of the left side of the mandible and upper portion of the neck. The patient stated that three days after the removal of a pyorrheic tooth six months earlier, slight swelling and trismus were experienced at the angle of the jaw. Hot wet dressings applied at the time gave no relief. The swelling continued to spread until the patient became seriously affected. Eleven days after the extraction, he was referred to a surgeon, who incised the swelling at the angle, releasing a large amount of pus. Drainage continued for about a month. The patient was then dismissed, although some swelling and trismus persisted. These symptoms later became severe from time to time, but subsided with regular application of hot wet dressings. It was only after they failed to be alleviated that the patient came to the clinic for treatment.

Examination.—There was no pain. The temperature was normal. Blood and urine examinations were negative. Roentgenographic examination of the involved area disclosed the absence of the mandibular second biscuspid, but no evidence of osseous infection.

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Course.—The patient was kept under observation for a week, during which time the surface of the skin covering the affected region developed several parallel rodlike elevations. Each elevation promptly softened and gave rise to an abscess, which was incised and drained.

Diagnosis.—Sulfur granules contained in the discharge were examined under the microscope, but gave no evidence of the flower-like colonies of the actinomyces observed in Case 1. The presence of gram-positive mycelia identified by the bacteriology department of the university led to the diagnosis of actinomycosis. The patient confessed to a habit of chewing straw.

Treatment and Outcome.—On establishment of the diagnosis, the lesions were irrigated with fresh lymph gland extract as previously described. Complete recovery was obtained twenty-six days after institution of the treatment.

COMMENT

Though actinomycosis may be rare as compared with other infections of the jaws, it is very likely that it would be found more frequently if every case of facial and cervical abscess were more carefully diagnosed.[3] According to Padgett,[4] about 60 per cent of persons affected by the disease have lesions involving the head and neck. In from 15 to 20 per cent, the disease follows the extraction of teeth. In Case 2, symptoms arose immediately after extraction of the left second mandibular bicuspid, but actinomycosis may have existed in a quiescent phase before the operation. The true nature of the "pyorrheic" condition of the tooth is unknown.

The etiology of actinomycosis is controversial. Lord[5] demonstrated that the actinomyces is frequently found in carious tooth substance and in the crypts of the tonsils of actinomycosis-free persons. It is also found on straw and grass, but it is possible that these fungi, commonly suspected of being pathogenic, may be aerobic and innocuous.[6] Nevertheless, both cases had a background of habitual straw chewing or picking of the teeth. It is conceivable that a practice of this kind might traumatize the oral mucosa or gingival tissues and provide entrance for the actinomyces either in the mouth or on the straw. However, the habit as a causative influence is minimized by the fact that many persons chew, and pick their teeth with, straw, but comparatively few develop the disease.

A study of the signs and symptoms discloses that the regional lymph nodes were not involved; that pain was very mild or entirely absent, and that the lesions were characterized by rapid proliferation of fibroblastic elements causing early stiffness, induration and trismus, followed by corrugation of the skin surface due to abscess formation and scarring in later phases. The course of the disease was chronic, with intermittent stages of subacute or acute activity. When suppuration occurred, spontaneous drainage often took place through sinuses in the granulation tissue. Contracted scars persisted after healing of sinuses. Trismus continued throughout the course of the disease. It may be considered a protective mechanism against

spread of the infection. Forceful opening of the mouth under general anesthesia may have a severe inflammatory reaction.

Positive diagnosis in incipiency is difficult or may be impossible for lack of distinguished criteria. Although characteristic sulfur granules usually can be found in the discharge of freshly opened abscesses, suppuration takes place only in later stages. When it has persisted for some time, granules may not be detectable. The surgeon who first incises the lesion seems to have the best opportunity for diagnosis.

Colonies of the actinomyces are at times seen in biopsy, but large numbers of sections may be made before a colony appears. Lymphocytes, histiocytes, plasma cells and mononuclear leukocytes surround the colonies during chronic stages, but polymorphonuclear leukocytes predominate in the subacute or acute phases. According to Siegmund and Weber,[7] an abundance of foam cells is an indication of actinomycosis. However, culture of the micro-organism provides the best basis for diagnosis. When clinical findings indicate the disease, but the fungus cannot be found, it is wise to retain the clinical impression pending further and repeated attempts to isolate the organism.

Differentiation of actinomycosis from common septic lesions, tuberculosis, syphilis and neoplasms is important. Tuberculosis is easily distinguished by the presence of tuberculous giant cells in biopsy material. Gummas differ from actinomycotic lesions by their typical punched-out appearance, indurated base and more rapid progress. A further criterion is available in the application of the Wassermann test. Various neoplasms are most readily distinguished through their histopathologic characteristics. Although useful to some extent in the differential diagnosis of lesions involving calcified tissues, roentgenographic findings in cases of actinomycosis offer no distinguishing characteristics.[8]

Adequate incision and drainage are as important in the treatment of actinomycosis as they are in other septic infections. Though used with success by others,[9] potassium iodide was not found particularly helpful in our hands. The same is true of sulfanilamide, successfully employed by Dobson, Holman and Cutting.[10] Sulfapyridine, sulfathiazole and penicillin were not available. It is conceivable that these agents may be effective, especially in the treatment of secondarily infected cases. Radical excision is unduly disfiguring. Irradiation, which appears to be quite satisfactory,[11] was not available in cases treated at Chengtu.

Crude lymph gland extract appears to be particularly applicable to localized lesions. The exact mechanism by which the extract exerts its effect is not known, but its use is founded on the observation that regional lymph nodes are not involved in actinomycosis.[12] Such observations would indicate that lymphoid tissues contain a factor that is detrimental to the growth of the fungus. More extensive clinical and experimental data are

needed to explain this phenomenon and to improve the method of treatment.

SUMMARY

A crude extract of the cervical lymph glands of goats has been found useful in the treatment of cases of chronic actinomycosis. Adapted particularly to local administration, application of the extract to diseased tissues appears to prevent the growth of the actinomyces and permit rapid healing in the absence of secondary infection. The method is experimental, but potentially sound, and deserving of serious consideration.

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Penicillin in Cellulitis Without Bacteremia.—Penicillin was used in twenty-five cases of severe cellulitis without bacteremia. *Streptococcus pyogenes* was the organism isolated in thirteen cases. In nine, *Staphylococcus aureus* was the organism of infection. In three, the infections were with green-producing streptococci and Vincent's spirillum. The results were satisfactory in twenty-two of the twenty-five cases. Failures or doubtful results were obtained in three.—Herrell, Nichols and Heilman in *J.A.M.A.*, August 12, 1944.

CONSERVATIVE SURGICAL TREATMENT OF AMELOBLASTOMA OF THE JAWS

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AMELOBLASTOMA is but one of the many appellations that have been applied to the tumorous growths which may develop in the human jaw, and which, despite the fact that they have very definite characteristics, have provoked much controversy, chiefly with regard to their surgical treatment. The literature is replete with designations, each intended to embody an accurate description of the form or origin of the lesion. Adamantine epithelioma, cystosarcoma, adenosarcoma, epithelial odontoma, cystoma, cystic tumor, chorioblastoma, ameloblastoma, polycystic adamantine epithelioma, true ameloblastic tumor, cystic adamantinoma, adamantine carcinoma, adamantinoma polycysticum, adamantoma solidum, multilocular cyst and adamantinoma cysticum are used as synonyms to describe the same lesion. Some of these terms are frankly inadequate, others are misleading. The word "adamantinoma" is probably the most commonly used, but it can be criticized in that it conveys an idea of hardness, which is entirely contrary to the physical appearance or texture of the tumor. However, the term "adamantinoma" is derived from the Greek *adamis,* meaning "of the hardness of stone." Enamel being very hard and resistant, the enamel-forming cell has been termed an adamantoblast. In 1902, Borst introduced the now generally accepted "adamantinoma."[1]

While from this standpoint the term cannot truly be considered as misapplied, it is certainly not consistent with the conception that it should evoke in the mind of the student. He is prone to think of the growth as containing enameloid structures rather than being a soft or cystic mass, resulting from the perverted activity of specialized epithelial cells. For this reason, "ameloblastoma" is preferable, since it indicates origin without influencing the idea of form or content. As the tumor form is somewhat variable, a descriptive term based on origin is obviously more suitable. Most contemporary surgeons and pathologists also apparently prefer this term, as is evidenced by its wide usage in the more recent writings. "Churchill and Ivy, in an as yet unpublished survey of tumors of the jaw, prefer to call the tumor 'ameloblastoma,' the new name which seems to be appropriate, and may in time be generally accepted."[1]

Historically, this growth gained early and widespread recognition as a clinical entity, and it has been a constantly recurring subject in medical and dental literature throughout the world. It continued to arouse keen interest in the minds of surgeons and pathologists because of the surgical

problems involved, and because of the variable and complex histologic picture exhibited by the cellular elements that engage in the formation and composition of the growth. There are available, as a result of these wide studies, many data and statistics that are proving to be of great value in current research.

According to a statistical chart compiled by Robinson, from English, German, French and Scandinavian periodicals, and referred to by Barr, a study of 248 cases revealed the average age of patients in whom ameloblastoma was found to be 37.6 years. Among 379 cases (the total upon which this chart is based), the average age at the time of discovery was 30.1 years, while the average duration of the tumor was 8.5 years. The extremes of age in which the growth was reported were the fourth and the seventy-sixth year. (Carpenter and Thoma report a case of ameloblastoma developing from a radicular cyst at the age of 68 years.) The mandible

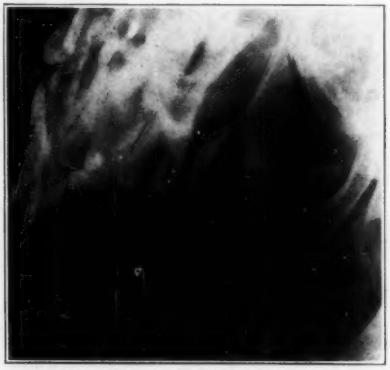

Fig. 1 (Case 1).—Preoperative roentgenogram, showing ameloblastoma characteristic of Group 1. (Multilocular type.)

was the site of the growth in 83.7 per cent of cases, and the maxilla in 16.3 per cent of cases. The sex apparently made no particular difference, the tumor occurring only slightly more frequently in females (54.3 per cent) than in males. In the matter of structural characteristics, 57.5 per cent of the growths were cystic; 24.2 per cent contained both cystic and solid parts, and 19.1 per cent were entirely solid. In an excellent paper by Kimm and Baranoff, published in the *Chinese Medical Journal,* the authors say, "It is known that adamantinomas are prone to occur in young persons, especially during the period of growth and development of the teeth."[2] This statement is essentially in agreement with my observations.

The shortest duration of the disease observed in their twenty-six cases was one-half year; the longest, eight years. According to the literature that

they examined, the shortest duration reported was thirteen weeks in a woman, aged 60. Swing and Albarran each have observed a case in which the tumor had been growing for thirty-eight years.[2]

The same authors quote Kegel as reporting five years after operation as the average period of time elapsed before recurrence. In one patient, it was eleven years. However, Albarran reported a case of recurrence eighteen years after a radical operation, and Porcelt one in which the recurrent growth appeared forty-five years later. Of Albarran's twenty-six cases, all but one lesion was of the multilocular cystic type. The average age was 31.1 years, almost that alluded to in Robinson's data (30.1 years), and of all jaw tumors, benign or malignant, that they observed over a period of approximately seventeen years, ameloblastoma constituted 7.4 per cent of the total—a much higher percentage than is generally accepted. Schroff states that "from a review of dental and medical literature, it appears that

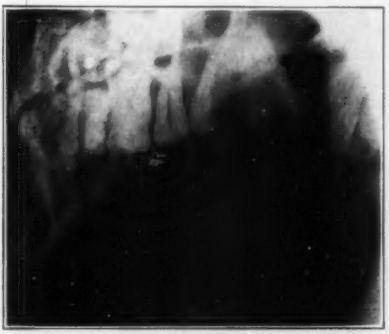

Fig. 2 (Case 1).—Postoperative roentgenogram showing extent of bone destruction.

adamantinomata occur rather infrequently." In a previous paper, he concluded:

> If every large cyst were examined microscopically, a great many more adamantinomata would be discovered. During the past year, in the Oral Pathology Department, I have examined 250 specimens of various mouth conditions consisting of tumors, cysts, granulomata and bone. Four adamantinomata were seen, all were from the lower jaw, three occurring in young women, ages 21, 26, and 29 years; the other, in a man aged 49 years.[3]

Bernier, in his review of thirty-four cases of ameloblastoma, states that, in the records examined, there were four cases of recurrence, following removal. Resection was carried out in three of these.[4]

SYMPTOMATOLOGY; DIAGNOSIS

Clinically, ameloblastoma may exhibit both subjective and objective

symptoms, which, however, cannot be considered pathognomonic. They may be indicative of any of several pathologic conditions which include ameloblastoma. Loosening of teeth, formation of fistulae or exudation, breaking through, degeneration of the mucous membrane, increase in size of the jaw at the site of the growth, change in facial contour, neuralgic pain and odontalgia may all be present in varying degree. On the other hand, ameloblastoma may be discovered accidentally by means of a routine roentgenographic examination, or even by a routine microscopic examination of a tissue specimen from an unsuspected lesion. While most recorded cases of ameloblastoma have resulted from disclosure of the growth by means of roentgenograms or clinical symptoms, or both, plus a subsequent histologic verification, it is certain, on the basis of observation, that a great many more cases have been diagnosed and operations performed than in some other form of lesion. This is often due to neglect on the part of the

Fig. 3 (Case 1).—Roentgenogram taken six years after operation; showing complete bone regeneration.

surgeon, who omits the microscopic examination. Such neglect, moreover, is not so much the result of carelessness as it is a direct consequence of the marked deviation in size and gross appearance of the lesion from the standard picture of ameloblastoma. While it is obviously not feasible to obtain laboratory verification on material taken from every case in which a surgeon removes pathologic tissue, although any unusual appearing tissue should be examined, it is essential if there is a history of recurrence or the area involved is in the lower third molar region and has, roentgenographically, a multilocular appearance; or when there is any doubt, however remote, as to the exact nature of the tissue. If, in such instances alone, this recommendation were followed, there would be fewer radical operations with consequent deformity, too often unavoidable concomitants in operations for recurrent ameloblastoma.

Roentgenographically, the appearance of this growth is that of a radiolucent area enclosed within a thick, heavy white line or border. While the radiolucent area may be homogeneous, with no loculation, as in the monocystic type, there is most often a division of the radiolucent region into numerous small, well-rounded chambers or cystic cavities of varying size, formed by the radiopaque walls or septa which traverse it; or it may present a moth-eaten appearance simulating osteitis fibrosa cystica. The white border surrounding the rarer monocystic form indents the radiolucent center with small radiopaque extensions, and the periphery may, in fact, be notched and lobulated.

Fig. 4 (Case 1).—Photo taken during operation, showing ameloblastoma removed from mandible.

The monocystic type is differentiated from the follicular cyst, with which it may be easily confused by the thin, smooth white circumference of the latter, there being no notching. The multilocular form has a general honeycombed appearance, the chambers exhibiting a characteristic roundness.

Further differential diagnoses based upon roentgenographic interpretation only involve osteitis fibrosa cystica (osteitis fibrosa) and central type giant-cell tumor. Greenfield states that "giant-cell tumors simulate adamantinomas, but these dark areas (cystic cavities) are not so well rounded,"[5] and that, as in osteitis fibrosa, the outline of the growth is not so sharply

defined. The traumatic cyst, incidentally, according to the same writer, appears as a well-defined radiolucent area, which may be irregular in outline and which has no white line about its periphery.

By and large, it may be said that the ameloblastoma almost always exhibits a definite heavy outline and a multilocular arrangement, the exception being the glandular type of ameloblastoma, wherein the bone is infiltrated, leaving little or no demarcations between diseased and sound bone, and resembles areas of osteitis fibrosa. Dentigerous, follicular and traumatic cysts, with rare exceptions, do not show divisions or pockets, but display

Fig. 5 (Case 1).—Photomicrograph, typical of Group 1.

a definite though thinner outline; while the central giant-cell tumor shows a single cavity with an indistinct outline.

PATHOLOGY

In gross or microscopic aspect, the ameloblastoma may be either cystic or solid, with some growths exhibiting both characteristics, and hence proving a more or less actively transitional type. The solid form consists

usually of amorphous nodular lumps of soft grayish tissue. The glandular type usually consists of hemorrhagic, friable, spongy bone, which can be cut with a knife, while the cystic type, which results from degeneration of the soft tissue comprising the solid tumor contains mucoid fluid, in which are found colloidal substances, the bone cavity being lined with epithelium. The central giant-cell tumor, on the other hand, consists of a soft reddish tissue resembling spongy liver, and is hemorrhagic.

The form and size of the bone cavity enclosing the tumor elements in ameloblastoma vary as to the stage in which its presence is disclosed. According to Waldron:

> The growth of the tumor is uneven and lobulated, and the bony cavity is similarly irregular. These characteristics are apparent in the roentgenogram. In more extensive tissue enlargements, the typical scalloped, multilocular appearance of bone destruction may be recognized. The fibrous capsular wall

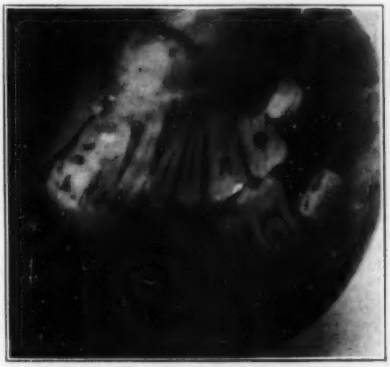

Fig. 6 (Case 3).—Preoperative roentgenogram, showing extent of bone destruction by the tumor (monocystic type).

> of an adamantinoma is quite adherent to the bone, and it would seem that small filaments of tumor tissue extend from the capsule into the bone. Recurrences are not infrequent, and thorough cauterization of the bony cavity walls is necessary. Excision of sections of the mandible is not necessary in the smaller adamantinomata.[6]

I have noted that invisible extensive infiltration of the bony walls in the cases operated on is most frequent in the glandular type. It is taken for granted, however, that invasive and infiltrative strands of tumor tissue may exist and may have penetrated the marrow spaces beyond the visible confines of the growth. Cauterization of the cavity walls is therefore practiced as a routine prophylactic measure.

Ameloblastoma is far more often encountered in the mandible than in

the maxilla, as previously noted. It usually starts in the molar region and spreads, by infiltration and erosion in the body of the mandible toward the symphysis, or in the ramus toward the sigmoid notch. Usually, if unchecked, it will proceed in both directions. As the tumor develops, it destroys the normal bone that lies in its path. It forms within itself locules or crypts, which may be connected by small tunnels, and which, as growth continues, tend to fuse owing to the thinning out and ultimate destruction of the bony partitions by the cystic degeneration which takes place. Clinically, ameloblastoma, if sufficiently large, may like other central tumors exhibit parchment-like crepitation upon palpation, as a result of destruction of the cortical plate of bone. In this respect, it differs from carcinoma, which never exhibits such a characteristic. Further differences between the ameloblastoma and the carcinoma aid in a differential diagnosis. Anesthesia of the lip usually indicates malignancy. Carcinoma occurs at a later age than ameloblastoma, and develops more rapidly. It also usually causes an

Fig. 7 (Case 3).—Postoperative roentgenogram.

impairment of the general health. The malignant form is most often accompanied by definite regional lymphadenopathy, which is absent in the benign form. Carcinoma will show mitotic figures and hyperchromatic nuclei upon histologic examination, and also evidences pleomorphism. Nevertheless, it must be remembered that, however rare or remote the possibility, there have been cases reported in which the ameloblastoma appears to have undergone metaplastic change into a malignant form or, at least, to have influenced either directly or indirectly the formation of a carcinomatous growth. This change is more likely to occur in ameloblastoma of the upper jaw. Bloodgood describes a rare jaw tumor which was completely removed from the mandible of a 15-year-old patient. It was recognized as an adamantine epithelioma or carcinoma by William Welch, professor of pathology at Johns Hopkins University. There were numerous recur-

rences, resulting eventually in loss of the entire left jaw. Six years before the patient's death, a nodule was discovered just in front of the ear, under the facial nerve. In an attempt to save the nerve, the growth was removed by blunt dissection. It was identified as an adamantine epithelioma—the same

Fig. 8 (Case 3).—Photomicrograph characteristic of Group 3.

type of lesion as the original tumor. There were three more recurrences, involving progressively the nerve, the gasserian ganglion and the brain. Death resulted from brain involvement.

"These adamantine carcinomas do not metastasize, but they may recur

when incompletely removed, and now and then we have accomplished a cure; even after recurrence."[7] Although this tumor originally, and in late recurrent form, was definitely established as an adamantine epithelioma, by an unimpeachable authority, such persistence and rapidity as evidenced in the history of the recurrences, together with the relentless progress of the growth elements upward through the soft structures, and eventually into the brain, constitute a picture entirely different from any of the ameloblastomas as described in the literature. Here, we see what appears to be a definite malignancy of the character of true carcinoma with no metaplasia involved at any stage. The case is a surprising exception, and the full history and histopathologic report, which were not included in the paper, should be interesting and valuable.

Fig. 9.—Preoperative roentgenogram of glandular type of ameloblastoma, Group 2.

Kimm and Baranoff, in the *Chinese Medical Journal*, state that "adamantinoma is locally malignant and exhibits a definite tendency to local infiltration and recurrence." They add that it metastasizes rarely.[2]

METASTASIS

There have been several reports of ameloblastoma with metastasis, and it is generally accepted that it may occur, but few writers produce histologic proof of such metastasis. Schweitzer and Barnfield report a case with histologic proof of metastasis to the lungs and pleura. The tumor started in the mandible, recurred locally after resection and spread to the maxilla, the calvarium and, finally, the lungs. The patient was subjected to twenty-four operations over a period of fourteen years, and aspiration of tumor cells into the respiratory tract was possible. The authors, however, believe that the tumor metastasized to the lungs by way of the blood stream.[8]

PATHOLOGIC GROUPINGS

Laboratory examination of the specimens which I have removed, or those which I have directly observed, indicates three distinct pathologic groupings: The first group conform to the more or less typical characterization of the growth found in the literature and show a classic "ameloblastoma" picture. The connective tissue stroma is studded with basal epithelial cells

Fig. 10.—Photomicrograph of glandular type of ameloblastoma (Group 2).

derived from the tooth anlage. These cells show all the differential changes found during the development of the enamel organ: basal, columnar (ameloblasts) and stellate cells, together with secondary cystic changes. Some may actually show a connecting link of basal cells between the tumor mass and the oral epithelium.

The second group, the glandular type, show vacuolated cuboidal and columnar ameloblastic cells without the stellate cells. Here, the cystic cavities are lined by the cuboidal and columnar cells and morphologically resemble a glandular structure. This type of ameloblastoma has a tendency

to infiltrate the bone, leaving no distinct visible demarcation between diseased and sound bone.

Those in the third group exhibit a distinct pattern, which may be delineated as an earlier form or precursive stage of the true or typical ameloblastoma. Herein are found all the histologic elements of a follicular dentigerous cyst wall. These are: a lining of squamous epithelium to the cyst and a fibrous connective tissue stroma that varies in density, degree of infiltration of chronic inflammatory cells; etc. At times, myxomatous degeneration may be noted. In addition to the foregoing structures, there are clusters and strands of epithelial cells within the cyst wall proper. They simulate the epithelial cells of the enamel organ, but are not so highly differentiated or so active as ameloblastic cells.

Fig. 11.—Roentgenogram of inoperable ameloblastoma of maxillae.

As regards the matter of the origin of the true or typical ameloblastoma, the theory of Malassez, advanced in 1885, continues, as always, to find general acceptance. The basic tenent therein is that the ameloblastoma (and the odontoma and supernumerary teeth as well) arises from epithelial rests or paradental débris, which, under certain conditions, becomes activated. The resultant cellular proliferation results in the formation of these anomalous structures. Local pressure or irritation (trauma); poor general resistance or malnutrition, and infection are all definitely significant in the etiology of ameloblastoma. Another important etiologic factor observed by many investigators is the unerupted or impacted lower third molar. The angle of the mandible is the most frequent site of ameloblastoma, or at least the area in which it originates. This is also, of course, the region occupied by the third molar. This fact, together with the ob-

served persistent retention of the developmental sac—which is usually large and thickened and not only surrounds the crown of the impacted tooth, but also is frequently adherent to the unerupted portion (usually the distal) of a partially visible tooth, plus the age, early adult life, during which the greatest incidence of ameloblastoma is reported, all point a finger of suspicion at this type of impacted third molar. As Goodsell observes, "It is entirely possible that these teeth (lower third molars) are in their buried positions because of the neoplasm, but one cannot help but wonder if there is an etiologic connection."[9]

The basic histologic description of the composition and growth of ameloblastoma is admirably set forth by Waldron[10]:

> The one characteristic common to all (ameloblastoma) is the presence of epithelium with marked intercellular bridges similar to the stellate reticulum of the enamel organ. There are also masses of epithelium which simulate the stratum intermedium, typical ameloblasts, and all states of transition between. The substance of the tumor is usually traversed by massive fibrous tissue trabeculae, which form the bulk of the tumor. The stroma may be very cellular. Masses of epithelium and broad anastomosing strands of epithelial cells are prominent characteristics of the adamantinoma. These epithelial masses undergo central degeneration and liquefy, leaving central cavities in which there is an accumulation of fluid. Areas of stellate cells with distinct intercellular bridges are found, while about the periphery of the cavities there is often a distinct layer of tall columnar cells resembling ameloblasts. In the early stages, the tumor is clinically solid. The cysts are then microscopic, and may on section so resemble a glandular structure that they have been misnamed adeno-adamantinum.
>
> As the fluid contents of the smaller cysts increase in amount, the intervening connective tissue stroma thins out, as does also the epithelial wall. Finally the degeneration of the septa leads to a breakdown of the walls, the tissue cells being added to the cyst contents. Larger and larger cysts may thus develop until one large cyst may simulate a dental root cyst.

Finally, a brief additional general description should help to round out the picture of this peculiar growth, and to make clearer the basis for the surgical technic here advocated by the author. It must be understood that ameloblastoma is a locally malignant central bone tumor which grows slowly by local infiltration of the marrow spaces within the bone, and which exhibits a strong tendency toward recurrence. While metastasis has been reported, it is extremely rare, and even if the lesion is metaplastic, the possibility of occurrence, however remote, is of vital importance as regards surgical procedure and prognosis.

My treatment of ameloblastoma utilizes a procedure based, first, upon certain principles derived from previous writings such as are alluded to in the foregoing material; second, on personal observation, and, third, on common sense. These main principles are three: 1. Unless there is complete destruction of the ramus or body of the mandible by the tumor,

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there is no attempt made to resect the jaw, so long as it retains its integrity of form and function and there is a matrix of normal bone left. There are those who feel that the osseous tissue immediately surrounding the growth should be removed to a safe depth (one-quarter inch has been suggested) as a safeguard against possible recurrence; and, if this depth should bring the operator to or beyond the external surface of the mandible, a summary resection should be carried out. I cannot subscribe to this view. Complete and careful removal of the tumorous material down to what, according to the visual and tactual senses of the operator, appears to be solid, healthy bone, followed by thorough chemical or electrical cauterization, is sufficient insurance against recurrence. The clinical results obtained from following this reasoning operatively have thus far, over a considerable period of years, and involving a fairly large number of cases, borne out this view. There have not been any signs of recurrence in any of the cases presented for periodic check up. Furthermore, were a growth to recur, it could again be removed, and were it, because of its extent, to require resection, the patient would have had the benefit of an interim—a matter usually of several years, according to the literature—during which he was entirely free of the social embarrassment, interference with function and psychologic damage that must attend facial deformity and disfigurement. As yet, there has been developed for these cases no prosthesis that is completely successful in circumventing all of these hazards.

2. For similar reasons, all operations for the extirpation of ameloblastomata are intra-oral. There is no advantage in resorting to the extra-oral approach, a method advocated by certain surgeons. The susceptibility to infection is no greater in the one case than in the other, provided ordinary precautions are taken. A qualified oral surgeon should experience no real difficulty in reaching and removing such a growth by operating intra-orally, no matter what region is involved. In fact, he would normally prefer such an approach because of his familiarity with the oral cavity, and because scars and all external markings could thus be avoided; a benefit to the patient of inestimable value.

3. To attempt to treat or reduce ameloblastoma by means of radiant energy, such as x-ray or radium, is usually unsuccessful. Ameloblastoma is usually radioresistant. This method of treatment should be employed only in inoperable cases.

A typical operation for the removal of an ameloblastoma may be described as follows: The mucoperiosteum is incised and retracted from a point about 2 cm. in front of the tumor margin, and is extended back sufficiently for complete exposure of the tumor mass. The thin buccal plate, if any, is removed, this exposing the growth. The consistency and type of the neoplasm is ascertained macroscopically. All solid and cystic portions of the mass are peeled out, the operator making certain that all crypts are exposed, and their contents carefully and thoroughly removed. The

bony limits of the growth must hence be seen and felt to be entirely free of cystic membrane or other foreign or pathologic tissue. The bone surfaces surrounding the growth may be painted with silver nitrate and then reduced with iodine or eugenol; or painted with phenol and neutralized with alcohol; or the entire wall may be fulgurized with the electrocautery. The cavity is filled with sulfathiazole crystals and gelatin, equal parts, and the mucoperiosteum is then replaced and sutured into position. No drains are inserted. Should the clot degenerate, the cavity is washed out at regular intervals until complete closure occurs.

Postoperative roentgenograms should be taken every four months for a minimum period of ten years, in order to make certain that complete bone regeneration has occurred, and there is no breakdown or recurrence. I have been able to follow over a considerable period of years twelve cases of ameloblastoma treated by conservative operation. No recurrence has been noted to date. The following cases have been selected as typical in the clinical and histologic aspects.

REPORT OF CASES

CASE 1.—*History.*—A woman, aged 32, robust and in good health, came to the office for treatment February 2, 1937. Two molars had been extracted from the lower right jaw about eight years earlier. The patient was told at that time that she had a badly impacted lower right third molar, which might erupt subsequently of its own accord. She had had intermittent pain in the same area for the past two years. A few months before her visit to the office, she noticed a swelling on the right side of the face and, intra-orally, on the right body of the mandible. She consulted a dentist and was referred to an oral surgeon, who told her that she had a tumor necessitating the removal of the right side of the jaw. The patient then sought council from a physician who specialized in that field, and he too advised the resection of the right side of the mandible. She then came to this office for an opinion.

Examination.—Roentgenograms revealed an impacted third molar on the inferior border of the mandible. The impacted tooth was surrounded by a large multicystic area beginning at the cuspid and extending distally to involve more than half of the ascending ramus, and reaching from the alveolar border to within one quarter inch of the inferior border of the mandible.

Operation.—February 5, the tumor and third molar were removed, under nitrous oxide-oxygen anesthesia. The walls were cauterized electrically by means of the bipolar current.

Outcome.—The patient made an uneventful recovery, and thus far there has been no recurrence. There is complete regeneration of the bone.

Laboratory Data.—The pathologic tissue was sent to Charles G. Darlington for examination, and the following report was received from him:

Macroscopic description: Large meaty tumor mass. At posterior pole and superior surface, transparent polypoid projections indicate cystic changes within the mass. These distended sacs give way to a firmer denser tissue, toward the inferior surface. At the opposite or anterior pole, the tissue is again cystic. The third molar tooth, which was unerupted, lies in a horizontal position

along the inferior surface of the cystic mass. The soft tissue which adheres to its cemento-enamel junction curls up toward the tooth's crown.

Microscopic description: Section shows a background of adult connective tissue. Impregnating this stroma are masses of epithelial cells which are clearly derivatives of the enamel organ. These masses show a limiting basal layer with cells at right angles to the basement membrane. These cells enclose a central core of starlike cells, linked together by delicate cytoplasm bridges. Frequently, this reticulum gives way to a keratin material. Here, the center of the mass becomes converted into a cystic space. At one pole of the slide, these epithelial masses are circumscribed, walled off by the "stroma." Here, the stroma simulates the wall of a cyst and the epithelial elements appear as polypoid masses lining and projecting into the sac.

Diagnosis: Adamantinoma.

CASE 2.—*History.*—A boy, aged 9 years, in good health, was being treated by an orthodontist, who noticed that the lower right second temporary molar was impacted, and that this impacted temporary molar was preventing the eruption of the second bicuspid. The patient came in March 11, 1942, to have the impacted temporary molar removed.

Examination.—Roentgenograms revealed that above the impacted temporary molar and the unerupted bicuspid was a radiolucent area resembling a small double cyst.

Operation.—Under nitrous oxide-oxygen anesthesia, the tissue overlying the two teeth was removed. Macroscopically, it was found to be soft, lobular and grayish, simulating an ameloblastoma.

Laboratory Data.—The tissue was sent to Dr. Darlington for biopsy, and the following report was given:

Macroscopic description: Folded and lobulated piece of soft tissue.

Microscopic description: Section confirms an evenly contoured piece of "soft tissue." Although the tissue mat is fibroblastic, its cellularity is not uniform. Wide areas are hyalinized. Here, cells are placed only at rare intervals and feeding vessels are just as rare. It is in these areas that the tissues are studded with trapped strands and clusters of the tooth germ; i.e. circumferential palisade of tall columnar cells and centrally placed reticulum. In some areas, the tissue takes a bluish stain. Here, intercellular matrix is nonfibrillar and seems to have undergone degeneration (myxomatous?).

Diagnosis: Ameloblastoma.

Second Operation.—April 2, the impacted temporary molar and the remaining ameloblastic tissues were removed. The bicuspid was left in position.

Outcome.—When last seen, June 28, 1944, the bone was completely regenerated and the bicuspid erupted into position.

CASE 3.—*History.*—A boy, aged 14, with a family history essentially negative, and who was normally developed and in good health, was referred to this office January 9, 1943, complaining of swelling and enlargement of the chin and lower anterior gum. The patient had first noticed the growth in the anterior portion of the mandible about three years before, and it was becoming progressively larger. When the lower anterior teeth protruded, he went to his dentist for consultation.

Examination.—Clinically, there was an enlargement of the anterior portion

of the mandible, both buccally and lingually. The lower anterior teeth were protruded and crowded out of alinement. The lower right cuspid and second bicuspid were not visible in the mouth. The mucous membrane was normal in appearance. Crepitation could be elicited on the buccal surface.

Roentgenograms revealed an extensive destruction of the mandible from the lower right to the lower left second bicuspid by what appeared to be a multilocular cyst. The lower right cuspid was impacted in the lower border of the mandible.

Operation.—January 9, the tumor and the teeth involved, as well as the impacted cuspid, were removed under nitrous oxide-oxygen anesthesia. The tumor was soft, amorphous, nodular and white, greatly unlike an ameloblastoma.

Laboratory Data.—The pathologic tissue was sent to Dr. Darlington for examination, and the following report was received:

Macroscopic description: 1. Large gelatinous and amorphous masses of soft tissue with one nodular area containing a hemorrhagic crater; lower right cuspid (unerupted) tooth. 2. Small irregular pieces of white friable tissue.

Microscopic description: 1. Several sections show basically a similar picture. This consists of an extreme myxomatous degeneration of loose fibrous tissue. This fibrous tissue is very vascular and is moderately cellular and in varying places shows a cellular infiltration. This infiltrate is composed of round cells, monocytes and even polymorphonuclears. Necrosis is also evident. One edge shows squamous epithelium. Another edge shows remnants of a fine layer of cells beneath which rare nests of cells of ameloblastic origin can be found. The latter findings suggest a cyst wall. 3. Section shows tab of fat.

Diagnosis: Follicular or dentigerous cyst with extreme myxomatous degeneration and potential ameloblastic transition.

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UMI

THE ADAMANTINOMA (AMELOBLASTOMA) ORIGINATING FROM THE IMPACTED LOWER THIRD MOLAR ENAMEL ORGAN

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BARRING some of the more formidable malignant lesions, it is undeniably true that the adamantinoma (adamantoblastoma, ameloblastoma or multilocular cyst) offers the least favorable prognosis of all

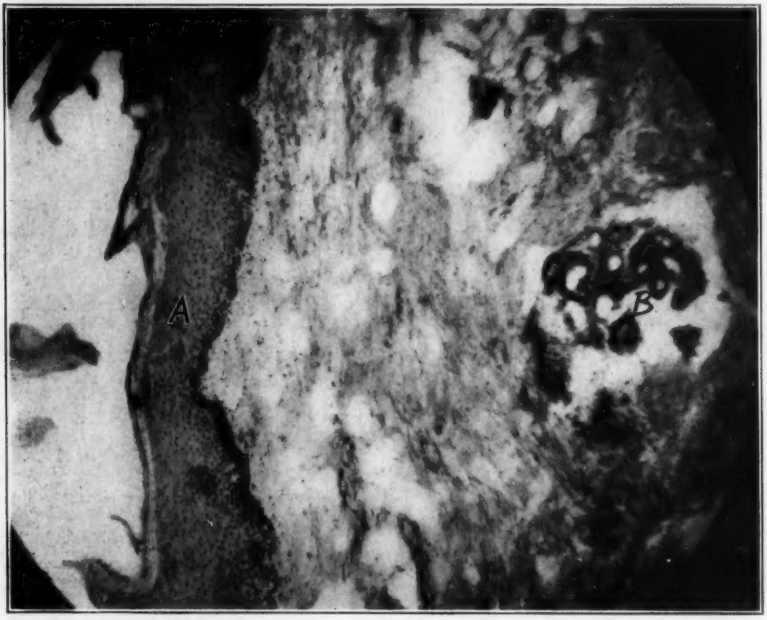

Fig. 1.—Low power photomicrograph of adamantinoma; showing abundance of fibrous tissue with epithelial cells distributed throughout. The epithelial cells have formed a definite layer at A. An island of epithelial tumor tissue is shown at B.

neoplasms that the oral surgeon may be called upon to treat. The high incidence of recurrence, with negligible amenability to irradiation, adds to the difficulty of surgical management. Even clinical observation of the most superficial sort seems to indicate the frequent association of the lesion with the enamel organ of the impacted lower third molar that has been either surgically ignored or incompletely removed, and, for this reason,

simultaneous consideration appears relevant. As available sources of reference are not in agreement concerning various phases of origin and treatment, it is believed that a brief review of authoritative opinion is indicated.

Microscopic description of the tumors is reasonably well standardized. Hertzler, describing their composition of epithelial-like cells and the fibrous tissue in which such cells are situated, mentions islands of giant-cell and squamous-cell tumor tissue[1] (Figs. 1 and 2). Thoma, in a more detailed discussion, recognized the epithelioma, stellate, ameloblastic and acanthoma types, in addition to adamantinocarcinoma, melanotic adamantoblastoma,

Fig. 2.—Another field of section shown in Figure 1; showing that distribution of epithelial tumor tissue has assumed definite cystic pattern.

hemangio-adamantoblastoma and adeno-adamantoblastoma.[2] In addition, derivation of the tumor is described as (1) a genetic disturbed enamel organ or tissue forming it; (2) cell rests of the enamel organ either during tooth development or later; (3) the epithelium of odontogenic cysts; (4) the surface epithelium covering the jaw, and (5) displaced epithelium in other parts of the body.[2]

Obviously, only the first three of the cited derivations are applicable to adamantinoma arising from the enamel organ of the impacted lower third

molar. Of these, the mention of cell rests of the enamel organ immediately suggests the débris of Malassez. Geschickter and Copeland attach little importance to the latter, stating that "the inner epithelial layer of the enamel organ is regarded as the site of origin for these growths rather than the outer and upper layer, which gives rise to the so-called epithelial débris."[3]

The adamantinoma is not considered as essentially a malignant growth, but, owing to its marked tendency to recurrence, it is sometimes described as locally malignant. Even this classification seems erroneous when it is recalled that metastases do not occur in its simple form; that the bony

Fig. 3.—Roentgen view of polycystic type of adamantinoma. The multiple cysts are well defined and lend a soap-bubble appearance to the tumor.

cortex is expanded and not destroyed except by pressure resorption, and that virtually all of the cells are mature. The extremely low incidence of mitotic figures explains its extreme resistance to irradiation.

In describing the roentgen appearance of the tumor, Pancoast, Pendergrass and Schaeffer say that "they appear as radiolucent areas with radiopaque lines throughout showing division in many compartments."[4] (Fig. 3.) That this is an accurate description of the polycystic type, or "multilocular cyst," is undeniably true, but, as the monocystic type is by no means rare, essentials of roentgen diagnosis must be mentioned.

Whereas the characteristic "soap-bubble" appearance of the polycystic type is virtually pathognomonic, a very real danger exists that the monocystic adamantinoma may be confused with an odontogenic cyst. Differential diagnostic criteria may be noted in that the cyst surrounds adjoining teeth, whereas the neoplasm is more apt to actually destroy their roots. This destruction is of the crisp, clean type that characterizes pressure resorption, instead of the irregular root erosion caused by the presence of pus in an abscess cavity. Furthermore, the outline of the cyst wall is usually smooth and regular; that of the monocystic adamantinoma is frequently characterized either by a notch in the cyst wall or by the nearby formation of

Fig. 4.—Monocystic type of adamantinoma; showing crisp, incisive pattern of destruction in first molar root structure. The third molar has been displaced to the head of the ramus. Several smaller accessory lesions are discernible.

smaller accessory lesions. Holmes and Ruggles[5] mention as part of its roentgen description the fact that the mass may contain calcified material. I am unable to corroborate this statement in other sources of reference.

It is generally agreed that, to be effective, surgical removal must be complete, if not radical. The mutilation occasioned by complete resection that destroys the continuity of the mandible warrants the deferral of the procedure to a status of last resort. Blair and Ivy mention the implantation of radium after enucleation.[6] Quick also mentions it and, in addition,

recommends heroic preoperative roentgen therapy.[7] Kaplan[8] calls attention to the resistance of adamantinoma to irradiation, but suggests that the surgical procedure be followed by postoperative roentgen therapy either alone or combined with radium according to the individual case. At first glance, the advisability of irradiating a tumor almost wholly composed of mature cells may seem debatable, if not inconsistent, yet normal discretion demands that a lesion of such intractability be given the advantage of all therapy that may be of any possible benefit. Such dosages must not be of sufficient intensity to cause severe irradiation osteitis.

It is believed that the high incidence of adamantinoma arising from the capsule of the impacted lower third molar warrants consideration of the site from this aspect. Mention has been made of predisposition of the areas to cystic disease,[9] and the epithelium of odontogenic cysts is accepted as a likely parent tissue. It logically follows that any lower third molar that is to be retained should be checked at regular intervals to determine whether any significant changes have developed in the roentgen image of the enamel organ. Any enlargement that is suggestive of cystic disease should be deemed a sufficient criterion for prompt condemnation of the tooth; but even this is not enough. The enamel organ or "capsule" must be meticulously removed in its entirety, and the area thoroughly cauterized. Good surgical judgment demands that the tissue be microscopically examined by a competent pathologist so that postoperative control may be adequately planned.

The management of the established lesions necessarily is governed by the case at hand. It is believed that roentgen therapy offers a slight, though contestable advantage, and certainly can do no harm if not carried to excess. Kaplan recommends a technic of 200 kilovolts, 0.5 or 1.0 mm. of copper and 1 mm. of aluminum filter at a distance of from 40 to 50 cm. Daily, 150 roentgen units are given, until a total of 3,000 roentgen units has been reached. Treatment may be repeated in four weeks.[8]

As involvement usually is noted in the ascending ramus and the posterior portions of the body, sufficient surgical access for enucleation and curettage can be gained by the extraction of the involved teeth and an incision down the anterior body of the ramus. After completion, radium therapy may be desirable. Here, Kaplan advises tubes of 5, 10 or 15 mg. in strength filtered by 1 or 1.5 mm. of platinum and encased in rubber sacks. The tubes are placed within the cavity in such a way as to obtain uniform radiation and are allowed to remain for such a time as to reach a dosage of 1,000 to 2,500 mg. hours, depending on the size of the excavated area.

Postoperative roentgen examination of the area should be made at yearly intervals so that possible recurrence may not remain unrecognized.

SUMMARY

1. The enamel organ of the impacted lower third molar must be regarded as a potential parent tissue for an adamantinoma.

2. All dental impactions should be checked regularly for roentgen evidence of cystic disease.

3. Despite the histologic composition of mature cells, the best opinion holds that the roentgen rays and radium are of importance in the treatment of the lesions.

4. The incidence of recurrence demands that postoperative examination be repeated at yearly intervals.

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Surgical Mind.—The graduate student may acquire a thorough knowledge of the science and possess the technical qualifications and yet fail to display certain qualities of mind which can alone elevate him above the common level and make him a real surgeon, something more than science, experience, love of work. In that something lie the specific qualities of the mind that are inherent and born with the individual, that fit the surgeon for his vocation. It is in the seed that we must look for these qualities which endow the individual with the attributes of intellect and of the character that constitute the surgical mind.—Matas.

OBTURATORS: A REVIEW

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PATIENTS with palatal defects labor under handicaps that cannot be fully appreciated by those with normal palates. The intimate relationship of the anatomic structures forming and separating the nasopharynx and nasal cavity from the oropharynx and buccal cavity determines the speech pattern and the mechanics of deglutition. Any interference with, or lack of, this relationship results in disorganization and distortion of speech and eating habits that should be corrected even though there may be compensating factors in the deglutition of certain patients with congenital deformities. Correction can be obtained by means of obturators or surgical operation, as indicated. This paper will attempt to discuss obturators only.

An obturator(Latin, *obturare,* to stop up) is a disk or plate or plug, natural or artificial, that closes an opening. More specifically, from the dental standpoint, it is a denture that corrects an opening of the palate: soft, hard or both.

HISTORICAL REVIEW

The obturator was one of the earliest appliances to be used in the mouth. The first known investigator to definitely record the idea of an appliance so constructed as to close off a palatine defect was Alexander Petronius, in the fifteenth century, although, no doubt, various simple appliances were improvised previously by persons afflicted with this type of defect. These would probably include wads of cloth, leather and wax.

Ambroise Paré, in 1541, published the first definite description of an obturator. The type that he employed consisted essentially of a disk to fit over the defect, attached to a sponge placed in the defect, and which would swell when moistened by the surrounding secretions and thus hold the disk up. He also constructed a cuff-button type with revolving stud. (Fig. 1.)

The French were predominant in this field until the early nineteenth century. Contributors included Pierre Fauchard, Bourdet and Delabarre. Descriptions of the construction of and other literature on obturators then passed over into the English language. Some of the investigators were Snell, Rowell, Hullihen, White, Suersen (who introduced the velum portion of the obturator) and Kingsley, who did a vast amount of research work and developed a fairly complete technic for the correction of congenital cleft palate that was applicable to postoperative cases as well.

The twentieth century includes the following as valuable contributors: Ackerman, who has done outstanding work in rehabilitating cancer patients; Bulbulian; Calvin Case; Eby and Kazanjian, who did much

From Columbia University School of Dental and Oral Surgery.

restorative work in World War I; Fitz-Gibbon, who has done much valuable work in speech rehabilitation of cleft palate patients; Lowery; Mitchell, who was the inventor of a specific type of hinged appliance that included the artificial vomer; Olinger; Ottolengui, and Young. Progress has been principally in the form of modification rather than originality, since the principles of design were established by the early workers. However, investigators have from time to time injected new designs and principles of technic, especially in speech-correction appliances. The rapid advances that dentistry has made in the field of dental materials has made it possible to construct appliances that are lighter, more sanitary and more correct physiologically.

Fig. 1.—First definite illustrations of obturators (designed by Paré, 1541).

Fig. 2.—Palatal opening, result of surgical removal of neoplasm.

CLASSIFICATION

Obturators can be classified into two types; namely, those that correct acquired openings and those that correct congenital openings.

Obturators to Correct Acquired Openings.—Openings of the soft or hard palate may be the result of a number of conditions, including trauma, such as gunshot wounds and the piercing of the palate with some sharp instrument such as a fragment of a steering wheel; diseases, such as syphilis, tuberculosis, osteomyelitis and cancer; radium and x-ray burns, and surgical operation, wherein portions of varying size are removed from the palate by the surgeon. (Fig. 2.)

The defects produced by the various above-mentioned conditions may vary in size from less than a centimeter in diameter to an area that may

include most of the hard palate, alveolar ridges and floor of the nose. Therefore, the periphery of the defect may be of bone, soft tissue or bone and soft tissue, and it may, of course, be anywhere on the palate.

This type of obturator may be constructed as a temporary or as a permanent appliance. The temporary type attempts only to close the opening and does not endeavor to restore the appearance. It does not include any teeth in its construction and may have stainless steel or gold clasps in dentulous cases. (Fig. 3.) It is made to serve only for an indefinite

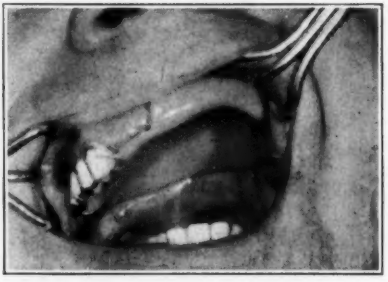

Fig. 3.—Temporary obturator. Acrylic and stainless steel clasps were used.

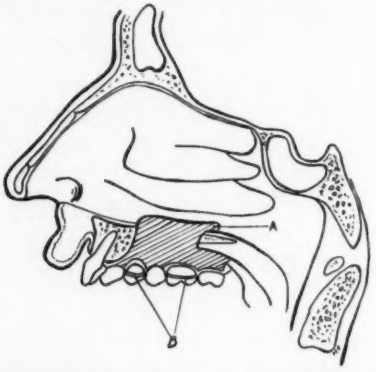

Fig. 4.—Permanent obturator to correct an acquired palatal opening in position. The means of retention are the posterior flange (A), which rests on the superior surface of the posterior border of the defect, and clasps (B).

period, but should not interfere with the regeneration of healing tissues, being indicated when the patient is undergoing prolonged treatment or the physician or surgeon believes that there is a possibility of recurrence of the disease. The obturator enables the patient to eat without having the food come out through the nose and to speak intelligibly, thus improving functional efficiency.

The permanent type, as its name implies, is constructed to last indefinitely. It consists of permanent materials, such as acrylic, chrome alloys or gold. It includes teeth to restore and improve functional occlusion and the appearance. (Fig. 4.) Speech is also improved. It is indicated when treatment has been concluded and the possibility of further surgical intervention in the wound area is remote.

Obturators to Correct Congenital Openings.—A cleft palate is a congenital fissure of the palate caused by arrested development of the associated developmental processes. Cleft palate may be divided into six types,

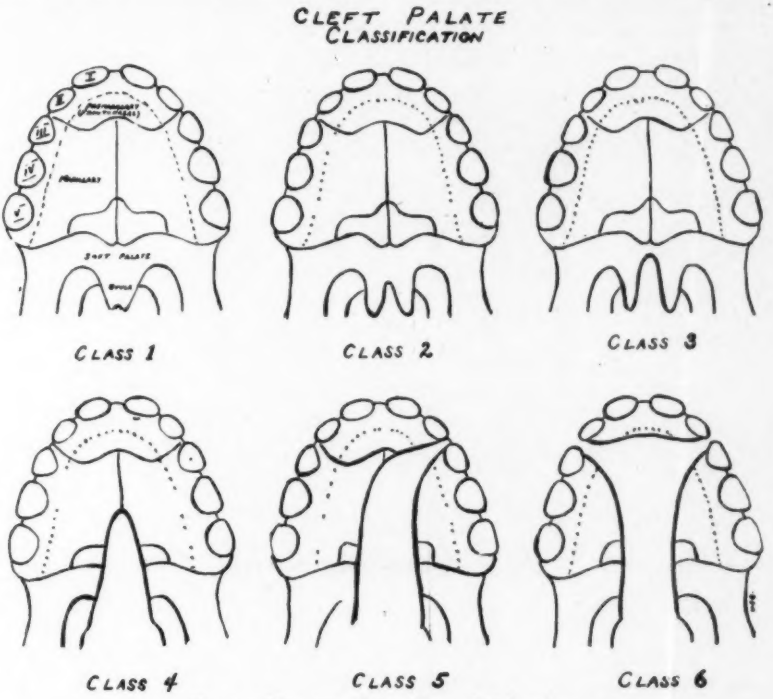

Fig. 5.—Diagrams of six classes of cleft palate.

depending on what portion of the palate is developmentally defective (Fig. 5.):

Class 1: Fissure of the azygos.
Class 2: Fissure in the uvula.
Class 3: Fissure in the soft palate.
Class 4: Fissure in the soft and hard palate.
Class 5: Fissure in the soft and hard palate through the alveolar ridge,

at the junction of the maxilla and premaxilla, usually accompanied by a cleft lip on the same side.

Class 6: Fissure in the soft and hard palate through the alveolar ridge, involving both sides of the maxilla, freeing the premaxillary bone, and usually accompanied by a double cleft lip.

The classes can be further subdivided into the postoperative, the edentulous and the postoperative edentulous.

Harelip may occur without a palate fissure, but the alveolar process is often involved.

The first two types are of no great concern to the dentist since they do not inconvenience the patient. Usually, they are not detected except in a very thorough mouth and throat examination. They are of interest to the

Fig. 6.—Fitz-Gibbon speech appliance, from practical case. (Courtesy of John J. Fitz-Gibbon.)

medical man. The remaining four are the types in which treatment is sought.

One reason that these patients seek treatment is their abnormal speech. This is adequately described by V. E. Marriott:

> The most common oral anomaly intimately related to speech is cleft palate. The person with a cleft palate has a flat, nasal tone, and his voice may assume a harsh unmelodious and unpleasant character. The sounds of k and g are practically non-existent, as the soft palate, which normally touches the tongue in making these sounds, is missing. In clefts of the hard palate, the consonants, such as t, d, r, n and l, are not present or greatly changed, as their utterance

depends largely on the contact of the tongue with the hard palate. All sounds of s and z, f, v, p, b, t, d, k, g, sh and zh are nasalized. After a surgical repair, there may still be nasal resonance or a nasal snort, due to lack of elasticity of the soft palate.

However, it has been demonstrated that some patients with a velar insufficiency or complete loss of palate structure can produce a k or hard g sound that is denasalized and quite acceptable. This velar insufficiency may be the result of an abnormal growth ratio. Thus, even though a child may at first have an adequately repaired palate, as he grows older the anteroposterior osseous development is not normal and velar insufficiency will result. Other factors in this growth ratio are the development and the eruption of the permanent molars, and tonsillectomies that are performed

Fig. 7.—Movable or pinlock hinge obturator.

after closures of the palate. The velar insufficiency may be corrected by surgery or by a prosthesis.

Other reasons that prompt the patient to seek assistance are to facilitate eating and to improve the appearance. The later would be involved in such associated deformities as sunken upper lip and retroclusion.

Fixed Obturator.—One form of appliance to correct congenital cleft palate is the fixed obturator, which has the palatal portion attached rigidly to the velar portion. There are three main types; namely, the solid obturator, the Kingsley obturator and the Fitz-Gibbon speech appliance. The solid obturator closes the defect by fitting into the nasopharynx and, by its walls, maintaining contact with the divided velum at all stages of phonation and deglutition. The Kingsley type has a flange contacting the divided velum, the portion attached to the plate being made of soft

vulcanite rubber. The Fitz-Gibbon type is an all-metal appliance composed of a palatal plate, a tail-piece that corresponds to the normal soft palate at rest and a bulb at the tail-piece whereby palatal function is simulated. (Fig. 6.)

Movable Obturator.—The other type of cleft correction appliance is the movable or pinlock hinge obturator. This appliance is composed of the palatal portion, pinlock hinge and artificial velum. (Fig. 7.) A vomer

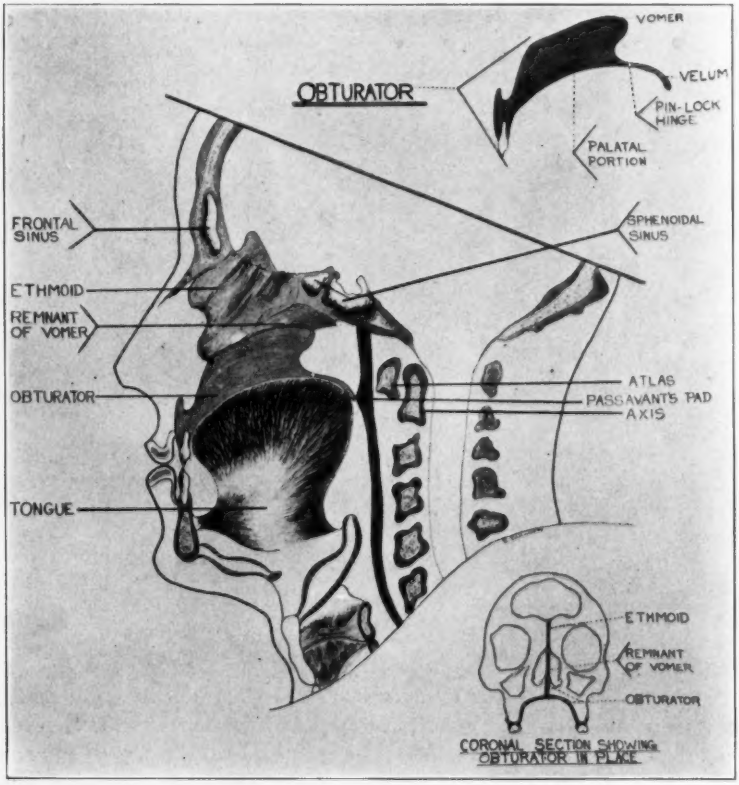

Fig. 8.—Median sagittal section of head showing obturator with vomer and "mobile" velum connected by pinlock hinge. Tissues and velum are shown in the act of deglutition.

portion may or may not be included. The movable type is indicated only where there remains an adequate flexible palatopharyngeal musculature.

This type is further subdivided into the temporary type and the permanent type. The former is constructed for children, whose jaws are changing

and who have few if any permanent teeth for clasping. In this case, the appliance consists of the palatal portion and clasps, and must be adjusted or renewed from time to time while the jaws grow. After development has stopped, a permanent type may be constructed. This consists of the above-mentioned movable type parts that close off the two cavities in speech and deglutition, the velum being hugged and lifted by the remnants of the functioning palatal muscles until its posterior border contacts Passavant's pad. (Fig. 8.) (Passavant's pad is a cushion of superior constrictor muscle usually formed on the posterior pharyngeal wall at about the level of the tubercle of the first cervical vertebra, or atlas. It is formed in the act of deglutition, etc., by the contraction of the palatopharyngeus muscle in pulling up the superior constrictor muscle to cause it to "bunch up.")

TECHNIC OF CONSTRUCTION

Obturators may be constructed from any of the accepted denture ma-

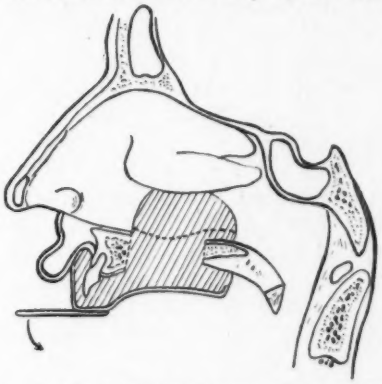

Fig. 9.—Impression in position. The dotted line indicates where the excess compound is later trimmed away.

terials; namely, acrylics, metal (gold or chrome alloys) and vulcanite. The temporary type usually is made of the more easily worked materials, since it is to serve for a limited time only. Clasps may be either wrought or cast.

Impression Taking.—Acquired defects: In cases of this kind, impression compound usually serves the purpose very well. When it is supplemented by an impression paste, an accurate impression of the defect and denture supporting structures is obtained in edentulous cases. In dentulous cases, the technic may be continued one step farther when cast clasps are to be constructed. Here, the compound impression of the teeth is cut out, hydrocolloid added and the whole impression put back in the mouth. The accurate detail of the teeth needed in surveying and designing the cast clasps is thus obtained.

Softened modeling compound is placed in a tray of adequate size and the combination placed in the mouth and pushed into place. If there is any danger of undercuts locking the impression, the anterior portion of the tray is moved down and up a few times (the posterior flange border being the fulcrum) while the compound is still soft, so that the undercut-locking compound portion is eliminated by dragging. (Fig. 9.) The impression material is then allowed to harden and is removed. Impression paste is next added to the compound surface in a thin layer and the impression is put back in the mouth, where it is kept until the paste sets. The additional step for cast clasps may now be carried out. The excess compound (along the dotted line) is trimmed off. The final impression is then poured.

In very large defects, wherein the greater portion of the hard palate and all of the alveolar ridge except the tuberosities are missing, a special technic is used to obtain an impression of both the posterior flange area and the floor of the nose, since both must be used for the appliance.

Congenital defects: Fixed type. The solid type of this group is no longer used as a rule, because its very bulk defeats its purpose of restoring speech. An impression technic for this type will therefore not be discussed.

The Kingsley type impression technic, as described by the originator, uses plaster as the impression material. A tray that will cover all the teeth (or ridges), but that is not overly large, is selected. An extension of wax or compound is next added to the posterior border of the tray, reaching as far back as the ends of the uvula, and so positioned that the relaxed soft palate with its uvula does not touch it at any point. Plaster of Paris is placed on the prepared tray in sufficient quantity to fill the roof of the mouth between the teeth, extending over the apron in a thin film, the excess being the part that will take in the teeth. All the area that is desired in this impression is that of the teeth and adnexa, soft palate at rest and border of the defect. It is stressed that the soft tissues must be relaxed.

When an impression of the structures in the nasal cavity is also required, the described technic is supplemented by the following procedure: The cavity above the roof of the mouth is filled with soft plaster to the border of the fissure. Before this plaster sets, the previous palatal impression, the surface of which is covered with separating medium, is placed in position and held there until the nasal portion sets. Both portions are then removed, the nasal with the aid of tweezers. They are then repositioned together, separating medium is applied and they are ready for pouring.

Briefly, the impression technic for the Fitz-Gibbon speech appliance is as follows: A suitable tray is selected and the center filled with softened modeling compound. An accurate impression of the defect is taken (added to or trimmed off as necessary), the pharynx with the protuberance of the atlas being included. This core is chilled and again placed in the mouth, and an impression of the rest of the mouth is taken in compound with a tray of adequate size and shape. After chilling, the entire impression is

removed, the core put in correct position and the cast poured. Correction of the cast is now required, since the soft structures included in the impression were compressed. This is done by means of a baseplate the tail-piece of which (covering the cleft velum) is adjusted until it follows the curve of the soft palate at rest. This baseplate is then put back on the model and the latter corrected to the new position to produce an accurate

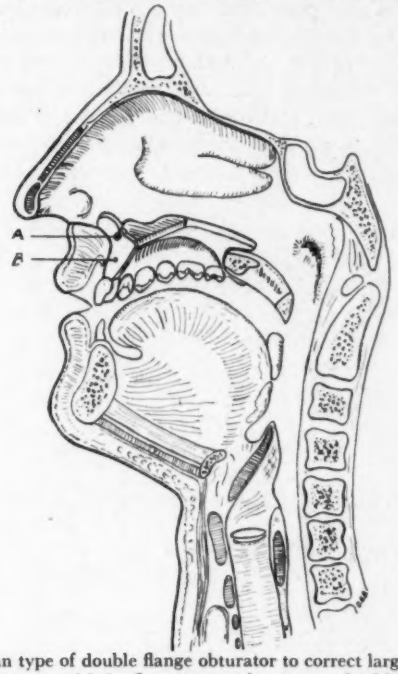

Fig. 10.—Ackerman type of double flange obturator to correct large acquired palatal defect. The anterior segment with its flange moves by means of a hinge (A) and is locked by means of a pin (B).

study model. The working model is obtained by using hydrocolloid and need not include the nasal cavity. It is poured in investment material directly, or in stone if a duplicating procedure is preferred. A master-mold technic is absolutely essential to finish the casting.

Movable type: The impression technic for obtaining the casts that are used in the construction of the pinlock hinge obturator is quite similar to that for the fixed type. Here also, the study cast includes the structures in the mouth and in the nasal and nasopharyngeal cavity, while the working model need include only the structures in the mouth and the periphery of the defect. The procedure is, first, to take an impression of the area above the cleft by means of compound on a small flat tray. The modeling compound is placed on the tray and the latter is put in the mouth and

gently pushed to place until the tray is about level with the cleft. After cooling, it is removed and trimmed or added to as required and softened in the necessary places, the procedure being repeated until an accurate impression is obtained. The tray is next removed from the compound and the latter replaced in the defect. A hydrocolloid impression is then taken of the remaining structures in the mouth. The two-part impression is then removed, assembled and poured in stone. The working model is obtained as follows: Softened compound is placed in the center of an impression tray of suitable size and shape and put in position in the mouth, the intention being to fill the defect. The compound around the border of the cleft is trimmed away, hydrocolloid is added and an impression of the rest of the mouth is taken. The compound thus prevents hydrocolloid from entering inaccessible parts of the cavity. The impression is then poured in stone.

Treatment of Casts and Models.—The casts and models, which are

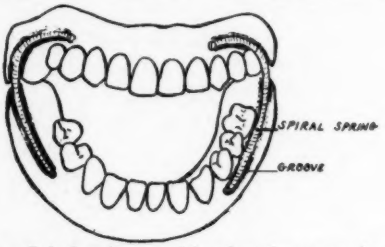

Fig. 11.—Spiral springs used in edentulous type obturators.

poured in stone, should be trimmed and made free of bubbles and excess plaster, to be ready for subsequent steps, including duplication, surveying and processing.

Design of Obturator.—The design of the obturators constructed to correct acquired palatal defects is relatively simple. Retention is obtained by means of the posterior flange and clasps in dentulous cases, and the posterior flange and peripheral and postdam seal in edentulous cases. In very large defects, added retention is procured by means of a flange which rests on the floor of the nose or fits into some undercut on the side and which works on a hinge principle to facilitate insertion and removal. (Fig. 10.) (Ackerman modification.)

Packing and processing are the same as for ordinary dentures.

Fixed type: The fixed type of obturator for the correction of congenital defects of the palate is retained by means of clasps in dentulous cases and spiral springs or a similar device in large-cleft edentulous cases. A greater number of clasps are required in order to distribute the stress of leverage action to as many teeth as possible.

Kingsley type: The Kingsley type will include the flanges and posterior

apron. The Fitz-Gibbon type also has the posterior extension. The latter type is an all-cast appliance.

Pinlock hinge type: The pinlock hinge type, because its stress of leverage action by the velum is dissipated in the hinge, usually requires only two or three clasps in dentulous cases and spiral springs in edentulous cases (where peripheral seal is inadequate). These springs are attached to the obturator by means of a peg, and to the lower denture also by means of a peg, or to a mandibular tooth or teeth by means of clasps. The spring fits into a groove in the obturator so that the bent spring, in its recoil action, tends to push against the obturator and thus hold it up. (Fig. 11.)

Construction of velum portion: In the fixed type, the procedure is to add a wire retention loop to the posterior apron and then to cover this with a bulk of carding wax. The latter is softened and the appliance is put in position in the mouth. The patient is then guided through a series of movements, including resting the chin on the chest, moving the head from side to side, swallowing and counting. The posterior portion is then studied to make certain that it includes the impression of the prominence of the atlas and that the lateral walls have been accurately trimmed by the palatopharyngeal muscles. The impression should be added to or trimmed as required and the process repeated until it is correct. A two-piece impression is then taken of the velum portion and the posterior part of the apron, for future correct positioning. The wax is removed, and the two-piece mold is used to make the wax form for a hollow casting or hollow acrylic bulb. The casting of the velar bulb is done separately, and then this portion is soldered to the palatal portion. The acrylic bulb is processed directly to the palatal portion.

The movable velum is constructed as follows: The female portion of the hinge is waxed to the posterior end of the palatal baseplate in such a position that the male portion, when in its uppermost position, will point toward Passavant's pad and will be centered correctly. The male portion is removed and the palatal portion, including the clasps and female portion, is processed. The male portion is reattached and a double thickness of baseplate wax is sealed to it. Its limits are the pharyngeal walls and Passavant's pad in the back. This wax is then softened and the entire appliance put in position in the mouth. The patient is then told to move his head forward and backward, from side to side, and to swallow. The appliance is removed and a little more than the excess wax is cut away. Soft carding wax is added to the velum wax and the appliance is again put in the mouth and the patient repeats the movements. The carding wax should make accurate contact with the palatopharyngeal muscles in their movements as well as with Passavant's pad, so that a perfect seal between the oropharynx and the nasopharynx is obtained. The last step is repeated until the seal is obtained. The pin locking the male portion to the female portion

is removed and the wax velum separated and then processed. The parts are then reassembled and put in position in the mouth.

PRECAUTIONS

After the obturator has been completed and inserted, the improvement in speech will vary in the different cases. Some patients will speak correctly in a very short time, while others will not be able to break themselves of incorrect speech habits unassisted. Therefore, it is essential to advise the patient to attend a speech clinic, where his problem will be correctly analyzed by competent teachers with experience in cases of this sort. It is also necessary for the patient to take certain mouth exercises to strengthen the palatopharyngeal muscles, in order that they may function properly in their sphincter action in conjunction with the velar portion of the obturator.

At times, an adherent upper lip must be freed by surgical measures and an adequate sulcus produced by the use of a stent.

SUMMARY AND CONCLUSIONS

Obturators should be constructed for patients with openings in the palate or with velar insufficiency. One of several types may be constructed, as determined by the enumerated indications.

The improvement in speech and eating habits after the obturator is inserted is well worth any effort involved. The patient is helped both physically and mentally.

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OSTEOPETROSIS WITH SUPPURATION AND EVENTUAL RESECTION OF THE MANDIBLE: REPORT OF CASE

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History.—T. B., a boy, aged 8 years, admitted to Cincinnati General Hospital, May 16, 1941, was kept under observation and treated for osteopetrosis (osteitis condensans generalisata; osteosclerosis fragilis generalisata; Albers-Schönberg disease; marble bone [so called because of appearance in x-ray film), bilateral cataracts, optic atrophy, hypertrophy of tonsils and adenoids

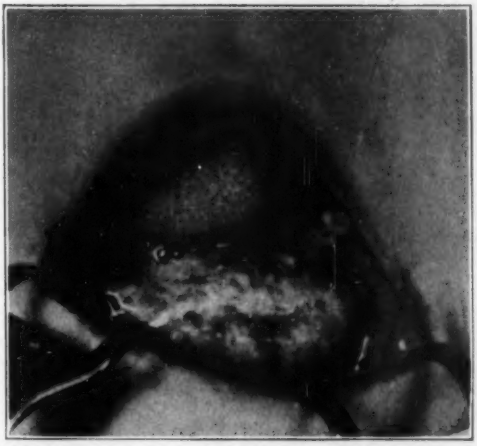

Fig. 1.—Appearance of ridge, showing necrosis, etc.

and dental caries of the deciduous teeth. The tonsils, adenoids and deciduous teeth were removed in May and the patient was dismissed June 10. He was admitted to the Cincinnati Children's Hospital January 6, 1942.

The patient was a full-term normal baby, weighing 9¾ pounds at birth. From the second to the ninth day after birth, he had jaundice. He was breast fed for twenty-two months. He was first given cereals at 6 months and vegetables and meat at 8 months. The diet was well supplemented with orange juice and vitamins, under a physician's care. The patient had three teeth at 4 months and all the deciduous teeth at 13 months, except the cuspids, which never did erupt. Growth seemed normal. The patient stood at 1 year, walked at 23 months and talked at an early age.

He entered the first grade at 7 years and then dropped out because of several operations for cataracts. At 8½ years, he was admitted to a school for the blind. The Hayes revision of the Binet-Simon scale for the blind showed the

Fig. 2.—Routine x-ray films (January 7, 1942).

Fig. 3.—Through-and-through drainage.

patient to be slightly dull, but not deficient. He was subject to colds, coughs and croup and was occasionally constipated. He had hemorrhoids when small. There was always impairment of vision and defective hearing. Cataract was diagnosed early. The patient had mumps when 1 year old, chickenpox,

Fig. 4.—Routine x-ray films (August 20, 1942).

Fig. 5.—Left lateral jaw plate after resection.

whooping cough and measles at 2 years. Vaccination and diphtheria immunization were given at school.

The family history was negative. The chief complaint was aching of the right jaw, with swelling and occasional fever, nausea and vomiting.

Examination.—The patient appeared dehydrated and apathetic. The skin was dry, hot and flushed. There was a complaint of swelling in the right mandibular area along the entire inferior border. Examination, externally, revealed a hard non-painful mass. Intra-oral examination showed that the oral mucosa was ruptured and the crest of the ridge was unlike the normal crest. (Fig. 1.) The ridge was denuded and hypertrophied. Purulent drainage was present.

Diagnosis.—The diagnosis considered was: (1) osteopetrosis; (2) osteomyelitis; (3) neoplasm, and secondary infection.

Treatment.—Hot magnesium sulfate mouth washes, sulfadiazene and rest in bed were prescribed. January 7, complete x-ray studies were made. The x-rays did not reveal a true picture of osteomyelitis, but only a picture of

Fig. 6.—Right lateral jaw plate after resection.

bone density. (Fig. 2.) January 12, under general anesthesia, the mandible was exposed intra-orally and a biopsy specimen was taken. Grossly, the mandible appeared to be necrotic and was hard and dark. The area was packed with iodoform gauze. The biopsy specimen showed dead, ragged bone with many polymorphonuclears on the surface. January 19, blood analysis showed: hemoglobin, 11.5 gm. per hundred cubic centimeters; red blood cells, 5,270,000; white blood cells, 14,800; 46 per cent polymorphonuclears; 36 per cent lymphocytes; 14 per cent monocytes.

January 22, an external incision, with drainage of the submaxillary area, was

carried out under general anesthesia. Through-and-through drainage was established. The patient received daily irrigations of potassium permanganate and sulfathiazole solution. The amount of discharge increased as the disease progressed. (Fig. 3.) March 12, the x-rays showed erosion and progressive loss of bony support of the teeth. March 19, it became necessary to extract the lower right molars and again establish through-and-through drainage. August 20, a routine x-ray examination revealed formation of a large amount of new bone. It appeared very dense and had irregular edges and seemed to completely surround the old bone. (Fig. 4.)

During the months of treatment, the blood picture varied, but showed a marked decrease in hemoglobin and red blood cells, with an increase in white blood cells. The hemoglobin dropped from 9 to 6 gm.; red blood cells, from 4,700,000 to 2,600,000. The white blood cells rose from 10,000 to 15,000.

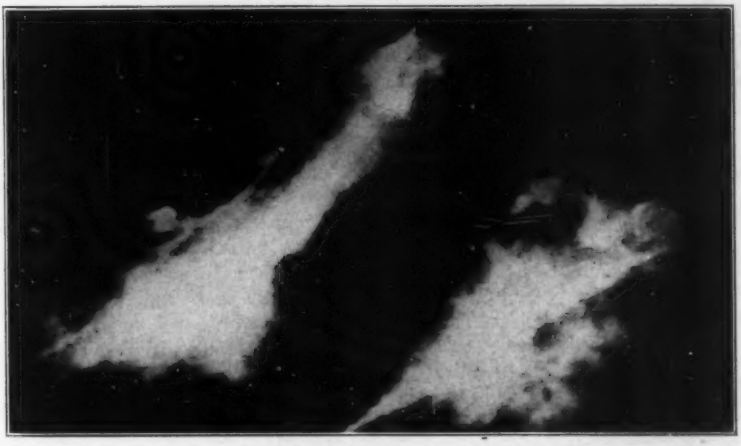

Fig. 7.—X-ray view of mandible after resection.

January 19, 1943, under general anesthesia, the mandible was resected and external incision, with drainage, was carried out. The mucosa was elevated and the mandible cut through bilaterally with rongeurs and the area packed with iodoform gauze. (Figs. 5, 6 and 7.) The patient had been receiving blood transfusions since January 1942.

Course in Hospital.—The patient's condition has steadily become worse. Sight and hearing have become much poorer than when he was first seen. The patient appears to be in good spirits in spite of the regression. It is interesting to note that, over a period of time, the head has become somewhat enlarged. The skull has become irregular and there are large protuberances and exaggerated frontal prominences. The cheeks appear to be swollen all the time, with an apparent decrease in size of the oral aperture. At times, it has been necessary to isolate the patient because of the exceedingly offensive

odor, due to suppuration. The progress of the disease gradually causes increased pressure on the optic and auditory nerves, with a decrease in these two functions. Prognosis is very poor. There is no known treatment at present and the suppuration can be treated only symptomatically. On resection of the mandible, suppuration ceased, the fistulas closed and the patient was discharged from the hospital.

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Pentothal Sodium Intravenous Anesthesia in Peace and War.—Pentothal sodium intravenous anesthesia has traversed the first ten years since its introduction. During each of these ten years, its use has increased generally throughout the country. Knowing that it is an agent of unusual anesthetic value, many of us have purposely overemphasized certain precautionary measures relative to its method of administration, dosage and selection of cases for its use. This attitude, we believe, is justified in teaching the use of a new anesthetic agent, the administration of which is so simple, the effects of which are so rapid and the results so satisfactory in most cases. The trouble has been that in the past many of the potential dangers incident to its use for certain patients and for certain operations have been overlooked or have been outweighed by its desirable effects. It is under these circumstances that untoward effects have occurred which at times have resulted in justifiable criticism of its safety. Pentothal sodium intravenous carries as wide a margin of safety as any established method of anesthesia if it is correctly administered and correctly applied. Those who have had a wide experience with the method have naturally found it a suitable method in more complicated surgical interventions than are generally considered to be within its scope. The most important advance has been the recognition of its potentialities when combined with other methods of anesthesia. These combinations have increased both the scope and the safety of the method. The value of the method in military surgery up to the present phase of World War II is more than gratifying. . . . There is still much to learn about intravenous anesthesia, and no doubt our present attitudes will continue to be modified from time to time. Perhaps new and structurally different agents will be evolved which will completely alter our present opinions of the method. Until such changes do occur, one has in pentothal sodium an intravenous anesthetic agent of undisputed usefulness, which has probably resulted in the most significant advance in anesthesiology throughout the last decade.—Adams, in *J.A.M.A.*, September 30, 1944.

UMI

ACUTE MENINGITIS AS A TERMINAL DISEASE RESULTING FROM PERICORONITIS ABOUT A PARTIALLY IMPACTED LOWER THIRD MOLAR: REPORT OF CASE

EUGENE V. LINSEY, A.B., D.D.S., Cleveland, Ohio

History.—A man, aged 23, first seen in the receiving ward on July 13, 1942, suffering from an acute toxic condition, was not able to concentrate and it was very difficult to obtain a coherent statement from him. Approximately ten days before, he had consulted a physician, because of a sore jaw and swelling in the submaxillary region. At that time, he was given "four white pills and a box of the same pills that were to be taken 2 every four hours." The box of pills that he had with him were 7½-grain sulfathiazole tablets. During the ten days, his condition had become progressively worse. In addition to the original symptoms, he had developed severe headaches and he complained that his eyes hurt and that his sight was impaired.

Examination.—There was a large edematous area laterally from and below the mandible on the left side. The swelling was firm, tender to touch and diffuse. The submaxillary gland was involved, as well as the superficial lymph glands of the neck. The patient was suffering with an acute pharyngitis, dysphagia, trismus, dehydration, exhaustion and general body weakness because of inability to take nourishment for the past week. The temperature was 102.5° F. It was difficult to make a thorough intra-oral examination. The lower left third molar was partially erupted, the crown being badly broken down and the pericoronal tissue inflamed. Slight pressure along the buccal mucosa released a steady flow of pus.

Postero-anterior and lateral left oblique jaw films were taken. The apical area of the lower left third molar showed a large rarefied area of bone. The patient was admitted to the Cincinanti General Hospital on a stretcher, and a preliminary diagnosis of acute alveolar abscess and cellulitis of pericoronal origin was made. Blood and urine analyses were ordered and fluids given intravenously.

Operation.—The next day, July 14, the patient was taken to the surgery. Premedication was given, morphine sulfate one-sixth grain and scopolamine one two hundredths grain. Before anesthesia, a nasotracheal tube was inserted. The operation was performed under nitrous oxide-ether anesthesia. The tooth was partially impacted, but very loose. An incision was made from the lingual distal angle of the tooth along the anterior border of the mandible for a distance of about 1¾ inches. The incision was carried anteriorly to a point between the first and second molars. The flap was then elevated free of the bone and held with an Allis clamp. Pus could be seen oozing from about the tooth. The tooth was easily removed and a pus pocket was encountered. The buccal mucoperiostium was elevated away and a second large

pocket with some pus was found. The lingual mucoperiostium was also elevated away and an iodoform drain was placed in the buccal pocket, in the socket and along the lingual aspect. The triangular flap of tissue was trimmed away and the socket left wide open, except for the above-mentioned drain.

Course.—Progress was accelerated during the next two days. The swelling subsided somewhat; the amount of pain and trismus lessened; swallowing became easier, and adequate drainage was maintained.

During the second postoperative day, the temperature rose from 101.1° F. to 104 and then quickly subsided, the patient resting more comfortably about two hours afterwards. He was sent to the surgery the next day to have the dressing changed and experienced a very heavy chill, which lasted about forty minutes. Supportive measures such as the use of heavy blankets, hot water bottles and hot tea, were employed at this time. Later in the day, there was another chill, and the temperature rose to 104.4°. A careful examination was made and the following conditions considered: (1) cavernous sinus thrombosis; (2) peripharyngeal or retropharyngeal abscess; (3) septicemia; (4) acute meningitis, and (5) involvement of the carotid sheath and thrombosis of the jugular vein.

The condition remained unchanged for a few days. The temperature then rose to 105° F. and the patient became stuporous and delirious. Examination showed a deviation of the eyes to the left and a slight left ankle clonus.

Second Operation.—July 21, an incision, with drainage, was made by the eye, nose and throat department, with my assistance. Intubation was resorted to before the anesthetic was administered and the operation was performed under nitrous oxide-ether. A midline incision was made from the upper border of the cricoid cartilage to the sternal notch. A tracheotomy was performed and a No. 6 tracheotomy tube was placed. Copious amounts of foul pus, blood and mucus were coughed up. An aspirator was used continuously and the bronchial tree was cleaned out. The remainder of the anesthetic was then administered through the tracheotomy tube. The area about the angle of the mandible was redraped and an incision was made just below the inferior border of the mandible at the angle. The anterior facial vein and angular veins were tied and severed. The submaxillary gland was pushed to one side and the peripharyngeal space was entered by blunt dissection. The styloid process of the pterygoid bone was encountered and a deep pocket of foul dark green pus was opened. Drains were placed and the patient was taken back to the ward. Oxygen therapy, started in the surgery, was continued.

A neurosurgical consultation was requested. Lumbar puncture was performed and the spinal pressure was found to be over 400 mm. Sodium sulfadiazine, 3 gm. every six hours, and repeated lumbar punctures every six hours, until the pressure was somewhat lowered, were prescribed.

Course.—After drainage of the parapharyngeal abscess, the condition seemed good for forty-eight hours. Although the patient was still unconscious, there was a drop in temperature, breathing became more regular and the amount of swelling lessened. During the late evening of July 23, the patient had a convulsion involving the right side, breathing became stertorous and there was a generalized twitching on the right side of the face, leg and arm. Avertin

(tribromoethanol) was given to control the convulsions. From this point on, the general condition of the patient became progressively worse, death occurring August 8. During the terminal period, the patient remained comatose, breathing at times was of a Cheyne-Stokes nature and convulsions were more frequent. Avertin was used continuously. Routine blood level determinations and urine analyses were made daily. A lumbar puncture was made as needed. Sulfadiazine was given intraspinally as well as intravenously. The patient received intravenous fluids as well as whole blood. When necessary, coramine was administered. Throughout the period, the patient was catherized regularly. The temperature rose very high during the terminal period and alcohol baths were used, the alcohol being vaporized with a large fan. Ice was also

MEDICATION AND CONDITION FROM JULY 21 TO AUGUST 5*

Date	Sulfa Level†	Pressure Mm.	Spinal Cells	Fluid Sulfa†	Avertin	Convulsion	Rectal Temperature	Pulse	Respiration
7/21	1.99	400	18,000		....	....	106.0	150	58
7/22	12.87	350	8,000	0.87	....	....	104.0	145	45
7/23	9.5	275	8,000	9.5	given	yes	105.0	120	60
7/24	11.8	375	6,000	8.4	given	yes	105.0	120	65
‡7/25	7.8	450	6,800	8.2	given	yes	105.5	110	50
7/26	7.2	150	....	....	given	yes	103.8	120	45
7/27	6.6	....	3,000	14.3	given	yes	104.4	125	45
7/28	7.5	....	....	....	given	yes	105.1	150	35
7/29	12.9	....	....	....	....	....	105.8	155	55
7/30	16.3	....	....	....	....	....	105.6	142	42
7/31	12.4	....	....	....	given	yes	105.2	128	43
8/1	....	....	....	....	given	yes	104.8	142	45
8/2	14.6	300	8,000	16.2	....	....	105.6	150	35
8/3	....	....	....	....	....	....	106.2	145	30
8/4	28.4	....	....	....	....	....	106.9	140	38
8/5	....	....	....	....	....	....	....	148	50

*The patient was continuously in coma. Blood culture showed streptococci after the first day.

†Milligrams per hundred cubic centimeters of blood.

‡The patient had a four plus Wassermann reaction and treatment was started.

packed about the patient on several occasions, to combat the high fever. Twelve days before death, a dermatologic consultation was requested because of a lesion on the external genitalia and a four plus Wassermann reaction was elicited. A diagnosis of chancre was made and treatment was started. At death, caffeine was used, to no avail.

Necropsy Report.—Necropsy revealed: (1) acute purulent basilar meningitis; (2) marked cerebral congestion and edema; (3) acute pneumonia of the right lower lobe and left upper and lower lobes, and (4) toxic changes in the kidney and the liver.

SUMMARY

This case shows the importance of timely diagnosis and initial treatment of

acute pericoronal infections. It is important that the condition be recognized and then treated by one who has a knowledge of the proper procedures. Early conservative treatment is the method of choice in combating pericoronal infection. This consists of daily irrigations, sulfa therapy, if indicated, and necessary supportive measures. Once the infected area is healed, the tooth can be extracted without likelihood of serious complications. Had penicillin been available, it might have been of service in the treatment of this patient.

310 Osborn Building.

Fractures of the Mandible.—Review of the recent literature confirms the authors' enthusiasm for skeletal fixation in selected cases of fracture of the mandible. It is easily applied, maintains immobilization and permits ready access to the parts for further reduction when needed. Complete function of the mandible is retained with a minimum of nursing care and no feeding or nutrition problem. There is no concern in these cases regarding nausea or vomiting. The technique for reduction of a displaced posterior fragment is simplified. The armamentarium of the oral surgeon is fortified so that he is able to treat a fractured mandible in the presence of a fractured maxilla. In extensive injuries of the face with loss of bony segments, external skeletal fixation is readily applicable for maintenance of space to facilitate later plastic or graft surgery. Tooth extraction can be done whenever indicated. Nevertheless, while there is now a tendency to use external skeletal fixation in many fractures which are amenable to other conservative forms of therapy, it is to be remembered that certain dangers are inherent in inserting pins into the mandible. Our experience would indicate that the results with interdental and intermaxillary wiring are quite good, particularly with regard to occlusion, and whenever possible these should be employed. However, external skeletal fixation should be reserved for: badly displaced fractures of the edentulous mandible, fractures posterior to the dental arch, multiple fractures involving both the mandible and maxilla, mandibular fractures wherein air or sea transportation or a condition such as bronchiectasis contraindicates closing the teeth in occlusion, comminuted fractures of the mandible with destruction of a bony section in which the fragments cannot be held adequately by dental splints, fractures through the area of the symphysis mentis (with comminution and displacement) which are not effectively fixed in the reduced position by dental wiring or arch bars, fractures with considerable displacement, those compounded into the mouth, and those with infection and local sloughing in which there has been delay in treatment. It should also be used when the opportune time for interdental or intermaxillary fixation has passed.—Wiesenfeld and Meadoff, in *Permanente Found. M. Bull.*, 1944, p. 49; abstr. *Surg., Gynec. & Obst.*, September 1944.

Current Literature

EARLY CARE OF FACIAL WOUNDS. Stanley A. Myers (MC) USNR.

The author classifies wounds of the face, as seen in the South Pacific area, as simple when they involve merely the soft tissue of the face. Complicated injuries, occurring more often, consist of injuries communicating with the oral cavity, paranasal sinuses or bony framework of the face.

Wounds of the soft tissues alone were usually clean on the patient's arrival, having been treated previously with a sulfonamide powder and left open. Definitive treatment consisted of irrigation of the wound with a warm saline solution, redusting with sulfathiazole powder and application of hot saline compresses. After several days, closure of the wounds was possible, and healing usually occurred without further incident. Wounds that were found to be infected on admission were usually those in which primary suturing had been carried out early. These were treated as any infected wounds, by reopening, drainage and application of hot saline or boric compresses. All patients received sulfathiazole orally and topically. Treatment of wounds complicated by injury to the mandible was given in conjunction with the dental department. The jaw was immobilized as soon as possible, while the external wounds were left open and treated by local application of sulfathiazole powder. Injuries to the floor of the mouth were often infected, with sloughing. Slough areas were excised and the mouth irrigated frequently with a warm saline or an aqueous azochloramid (chloroazodin) solution.

Fragments of facial bones, if still attached to the soft tissue, were left in place, loose fragments were removed and the wound was treated conservatively. These wounds were usually infected by contamination from communication with the oral cavity. Daily removal of slough and daily irrigations and hot fomentations were most effective. In cases of cheek defect, as soon as it appeared safe after clearing up the infection, the mucous membrane was sutured to the skin, to prevent further retraction of the skin. If enough tissue remained, the wound was closed. Patients with wounds involving the oral cavity were fed by means of a nasal cather. Some patients fed themselves by means of a bulb syringe.

Complications during the early period after injury were infection, mostly in cases with communication with the mouth, sinuses and nasal cavity, cleared up by débridement, hot fomentations and sulfonamide therapy; obstruction to airways, requiring tracheotomy, and hemorrhage in one case with a shrapnel wound of the submaxillary region and floor of the mouth, stopped by ligation of the external carotid artery.

Generally, facial wounds should be treated as infected for long enough to obtain a clean field, before closure is made. Attached bony fragments should be left in place and there should be as little débridement as possible.

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